DISSERTATION ON

"ANALYSIS OF FACTORS FAVOURING ACUTE KIDNEY INJURY IN SNAKE BITE PATIENTS"

DISSERTATION SUBMITTED TO

THE TAMILNADU DR. M.G.R. MEDICAL UNIVERSITY

In Partial Fulfilment Of The Regulations

For The Award Of The Degree Of

M.D. - GENERAL MEDICINE- BRANCH – I

THANJAVUR MEDICAL COLLEGE,

THANJAVUR - 613 004.

THE TAMILNADU DR. M.G.R. MEDICAL UNIVERSITY

CHENNAI - 600 032.

APRIL - 2015

CERTIFICATE

This is to certify that this dissertation entitled **"ANALYSIS OF FACTORS FAVOURING ACUTE KIDNEY INJURY IN SNAKE BITE PATIENTS"** is the bonafide original work of **Dr.DEYAGARASAN E** in partial fulfilment of the requirements for M.D Branch -I (General Medicine) Examination of the Tamilnadu Dr. M.G.R. Medical University to be held in APRIL - 2015. The period of study was from **2014 JANUARY TO 2014 AUGUST**

Prof. Dr.D.NEHRU, M.D.,DMRD,
Unit chief M-5,
Department of internal medicine
Thanjavur Medical college,
Thanjavur - 613004

Prof.Dr.P.G.SANKARANARANAN,M.D.,
Head of the Department,
Department of Internal Medicine,
Thanjavur Medical College,
Thanjavur - 613 004.

PROF.DR.P.G.SANKARANARAYANAN, M.D.,
DEAN I/C,
THANJAVUR MEDICAL COLLEGE, THANJAVUR – 613 004

DECLARATION

I, **Dr.DEYAGARASAN E**, solemnly declare that the dissertation titled dissertation on **"ANALYSIS OF FACTORS FAVOURING ACUTE KIDNEY INJURY IN SNAKE BITE PATIENTS"** is a bonafideworkdone by me at Thanjavur Medical College, Thanjavur during Janauary 2014 – August 2014 under the guidance and supervision of **Prof.Dr.D.NEHRU, M.D.,D.M.R.D,** Unit Chief M-5, Thanjavur Medical College, Thanjavur.

This dissertation is submitted to TamilnaduDr. M.G.R Medical University towards partial fulfilment of requirement for the award of **M.D. degree (Branch -I) in General Medicine**.

Place: THANJAVUR.

Date: **Dr.DEYAGARASAN E**
Postgraduate Student,
M.D. in General Medicine,
Thanjavur Medical College,

ACKNOWLEDGEMENT

I gratefully acknowledge and my sincere thanks to the Dean, **PROF.DR.MAHADEVAN,MS** Thanjavur Medical College, Thanjavur, for allowing me to do this dissertation and utilize the institutional facilities.

I am extremely grateful to **Prof.DR.P.G.SANKARANARAYANAN.M.D,** Head of the Department, Department of Internal Medicine, Thanjavur Medical College, for his full-fledged support throughout my study and valuable suggestions and guidance during my study and my post graduate period.

I am greatly indebted to **PROF.DR.D.NEHRU, M.D.,,D.M.R.D,** my Professor and Unit Chief, who is my guide in this study, for his timely suggestions, constant encouragement and scholarly guidance in my study and post graduate period.

I Profoundly thank my professors **PROF.DR.K.NAGARAJAN,M.D PROF.DR.S.MANOHARAN,M.D;,DR.C.GANESAN,M.D,AND DR.GUNASEKARAN,M.D D.M.,** (Registrar), for their advice, guidance and valuable criticism which enabled me to do this work effectively.

I would like to express my gratitude to **PROF.DR.D.RAJENDRAN, MD DM** Department of Nephrology for his immense help in the study which enabled me to complete this work and also permitting me to utilize the facilities available in the department.

My sincere thanks to assistant professors **DR.D.SEKAR,M.D,D.M Dr.KAVITHA,M.D.,**; **DR.K.MUTHUSELVAN,M.D,** for their motivation, encouragement and support. .

A special mention of thanks to all the patients who participated in this study for their kind cooperation without whom the study would not be possible.

I would like to thank my colleagues and friends who have been a constant source of encouragement..

Thanjavur Medical College

THANJAVUR, TAMILNADU, INDIA-613 001

(Affiliated to the T.N.Dr.MGR Medical University, Chennai)

INSTITUTIONAL ETHICAL COMMITTEE

CERTIFICATE

Approval No. : 076

This is to certify that The Research Proposal / Project titled

ANALYSIS OF FACTORS FAVOURING ACUTE KIDNEY INJURY IN SNAKE BITE PATIENTS.

submitted by Dr. E. DEVAGARASAN **of**

Dept. of GENERAL MEDICINE **Thanjavur Medical College, Thanjavur**

was approved by the Ethical Committee.

Thanjavur

Dated : .....

THANJAVUR MEDICAL COLLEGE

Secretary

Ethical Committee

TMC, Thanjavur.

turnitin

Digital Receipt

This receipt acknowledges that Turnitin received your paper. Below you will find the receipt information regarding your submission.

The first page of your submissions is displayed below.

Submission author:	201211214.md General Medicine DE...
Assignment title:	TNMGRMU EXAMINATIONS
Submission title:	ANALYSIS OF FACTORS FAVOUR...
File name:	DERRETATION_2_Autosaved_25.do..
File size:	54.11K
Page count:	106
Word count:	13,941
Character count:	77,740
Submission date:	24-Sep-2014 08:44PM
Submission ID:	449344460

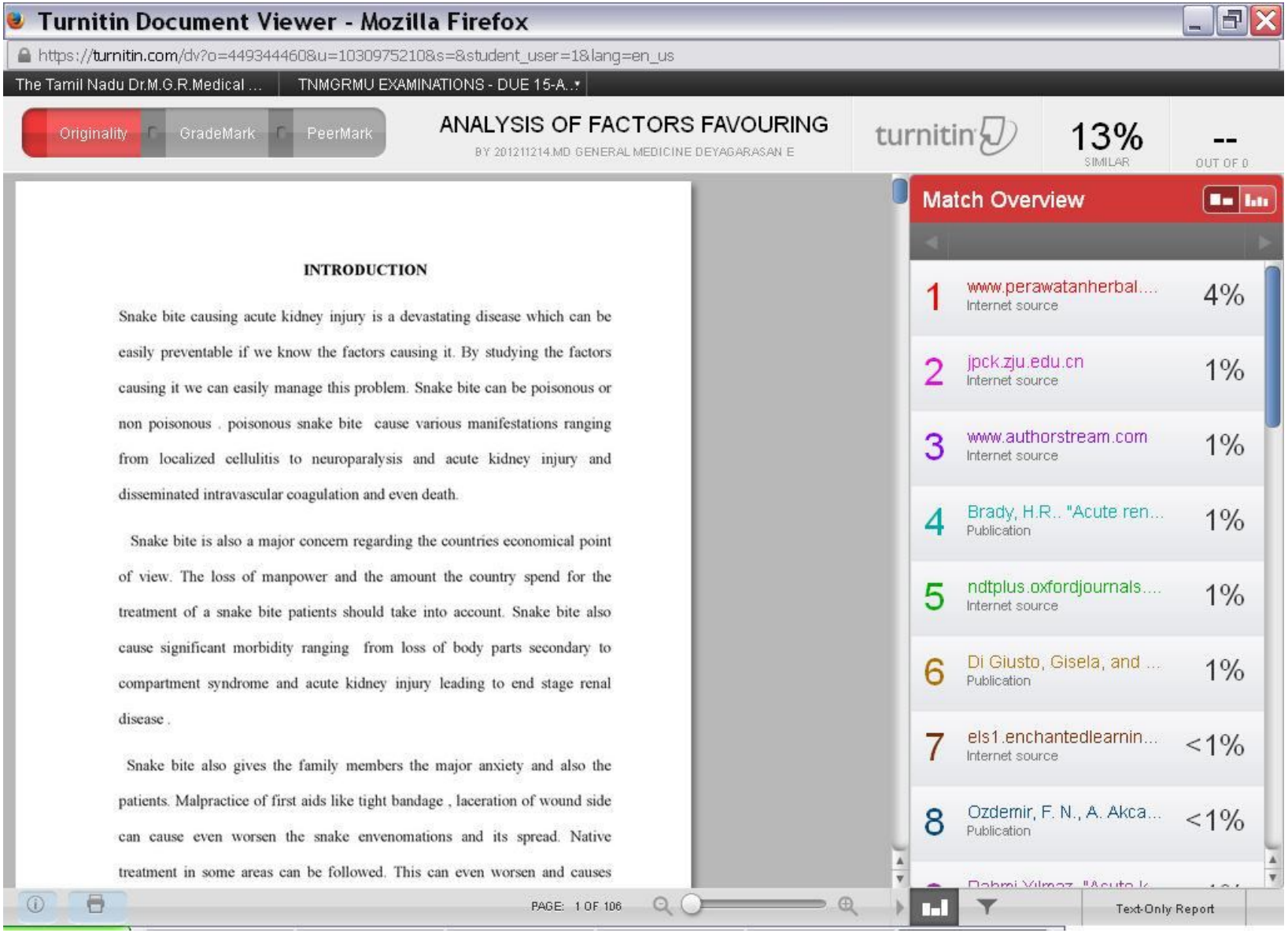
INTRODUCTION

Snake bite causing acute kidney injury is a devastating disease which can be easily preventable if we know the factors causing it. By studying the factors causing it we can easily manage this problem. Snake bite can be poisonous or non poisonous , poisonous snake bite cause various manifestations ranging from localized cellulitis to neuroparalysis and acute kidney injury and disseminated intravascular coagulation and even death.

Snake bite is also a major concern regarding the countries economical point of view. The loss of manpower and the amount the country spend for the treatment of a snake bite patients should take into account. Snake bite also cause significant morbidity ranging from loss of body parts secondary to compartment syndrome and acute kidney injury leading to end stage renal disease.

Snake bite also gives the family members the major anxiety and also the patients. Malpractice of first aids like tight bandage , laceration of wound side can cause even worsen the snake envenomations and its spread. Native treatment in some areas can be followed. This can even worsen and causes untoward side effects. Acute kidney injury in snake bites in most time is self limiting and reversible in correct time. Adequate hydration is the cornerstone of

1


Turnitin Document Viewer - Mozilla Firefox
https://turnitin.com/dv?o=449344608&u=1030975210&s=&student_user=1&lang=en_us
The Tamil Nadu Dr.M.G.R.Medical ...
TNMGRMU EXAMINATIONS - DUE 15-A..
Originality
GradeMark
PeerMark
ANALYSIS OF FACTORS FAVOURING
BY 201211214.MD GENERAL MEDICINE DEYAGARASAN E
turnitin
13%
SIMILAR
--
OUT OF 0
INTRODUCTION
Snake bite causing acute kidney injury is a devastating disease which can be easily preventable if we know the factors causing it. By studying the factors causing it we can easily manage this problem. Snake bite can be poisonous or non poisonous . poisonous snake bite cause various manifestations ranging from localized cellulitis to neuroparalysis and acute kidney injury and disseminated intravascular coagulation and even death.
Snake bite is also a major concern regarding the countries economical point of view. The loss of manpower and the amount the country spend for the treatment of a snake bite patients should take into account. Snake bite also cause significant morbidity ranging from loss of body parts secondary to compartment syndrome and acute kidney injury leading to end stage renal disease .
Snake bite also gives the family members the major anxiety and also the patients. Malpractice of first aids like tight bandage , laceration of wound side can cause even worsen the snake envenomations and its spread. Native treatment in some areas can be followed. This can even worsen and causes
PAGE: 1 OF 106
Match Overview
1 www.perawatanherbal.... Internet source 4%
2 jpck.zju.edu.cn Internet source 1%
3 www.authorstream.com Internet source 1%
4 Brady, H.R.. "Acute ren... Publication 1%
5 ndtplus.oxfordjournals.... Internet source 1%
6 Di Giusto, Gisela, and ... Publication 1%
7 els1.enchantedlearnin... Internet source <1%
8 Ozdemir, F. N., A. Akca... Publication <1%
Text-Only Report

CONTENTS

ABSTRACT

BACKGROUND AND OBJECTIVE

Snake bite poisoning is a major dreadful event to the human beings. The complications related to kidneys are observed in majority of patients with poisonous snake bite.This study is an attempt to study the various clinical profile of snake bite patients and evaluation of acute kidney injury in them.

METHODOLOGY

Fifty patients with snake bite induced acute kidney injury were selected randomly and their clinical profiles were analysed and AKI was evaluated using noninvasive methods.

RESULTS

Out of 50 patients in the study, coagulopathy was observed in 100% of patients, dehydration in 68 % , russel viper among the common species of 68 %, cellulitis in 52%, with no major advantage of giving high dose ASV and early institution of ASV.

CONCLUSION:

Coagulopathy is a major factor in causing aki followed by dehydration and russel viper bite. There is no major advantage in giving high dose ASV or early institution of ASV or cellulitis .

KEY WORDS: Snake bite ; coagulopathy ; dehydration ; cellulitis ; AKI ; ASV.

INTRODUCTION

Snake bite causing acute kidney injury is a devastating disease which can be easily preventable if we know the factors causing it. By studying the factors causing it we can easily manage this problem. Snake bite can be poisonous or non poisonous . poisonous snake bite cause various manifestations ranging from localized cellulitis to neuroparalysis and acute kidney injury and disseminated intravascular coagulation and even death.

Snake bite is also a major concern regarding the countries economical point of view. The loss of manpower and the amount the country spend for the treatment of a snake bite patients should take into account. Snake bite also cause significant morbidity ranging from loss of body parts secondary to compartment syndrome and acute kidney injury leading to end stage renal disease .

Snake bite also gives the family members the major anxiety and also the patients. Malpractice of first aids like tight bandage , laceration of wound side can cause even worsen the snake envenomations and its spread. Native treatment in some areas can be followed. This can even worsen and causes untoward side effects. Acute kidney injury in snake bites in most time is self limiting and reversible in course time. Adequate hydration is the cornerstone of

treatment. Timely administration of antisnake venom can also prevent the development of acute kidney injury and can retard the progression of disease.

There are many factors in snake bite can also favour the occurrence of acute kidney injury. These studies focuses the factors which are more prone for acute kidney injury.

REVIEW OF LITERATURE

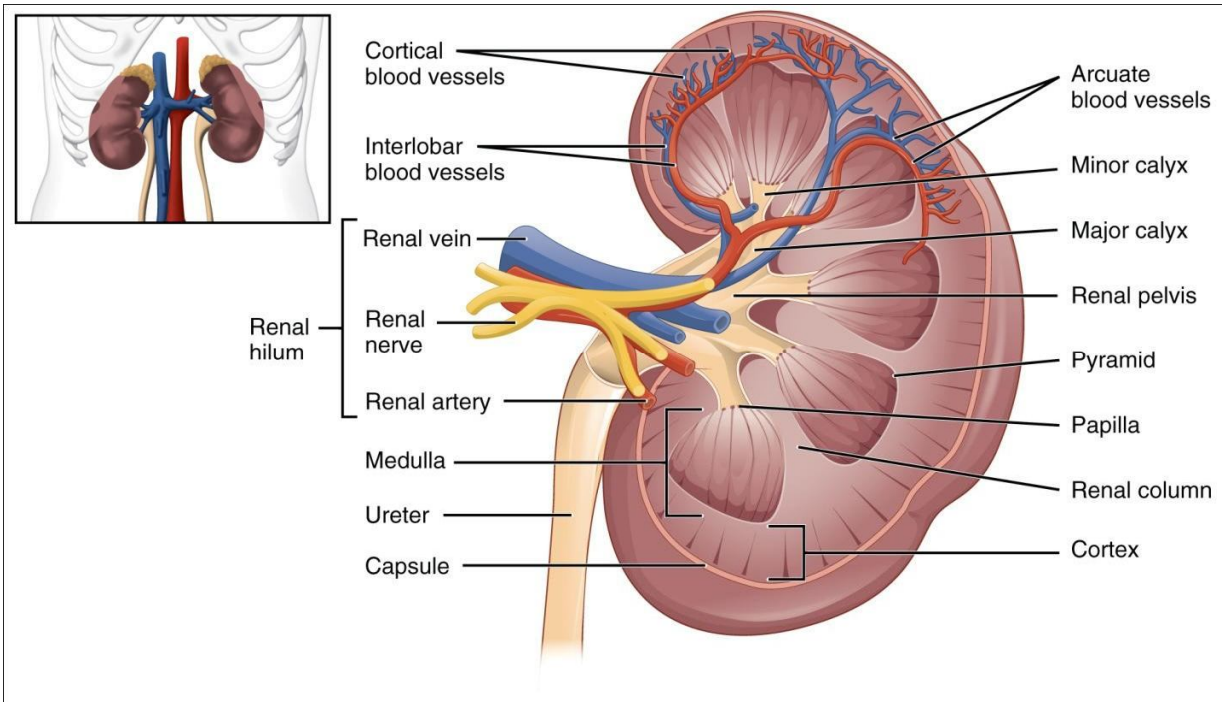
CLINICAL ANATOMY OF KIDNEY

Kidneys are paired retroperitoneal organs . they are situated in the posterior side of the abdomen. They are placed on each side of the vertebral column. upper pole lies opposite the 12^{th} thoracic vertebra. The lower pole opposite the 3^{rd} lumbar vertebra. The right kidney is usually lower and left kidney is higher in position.

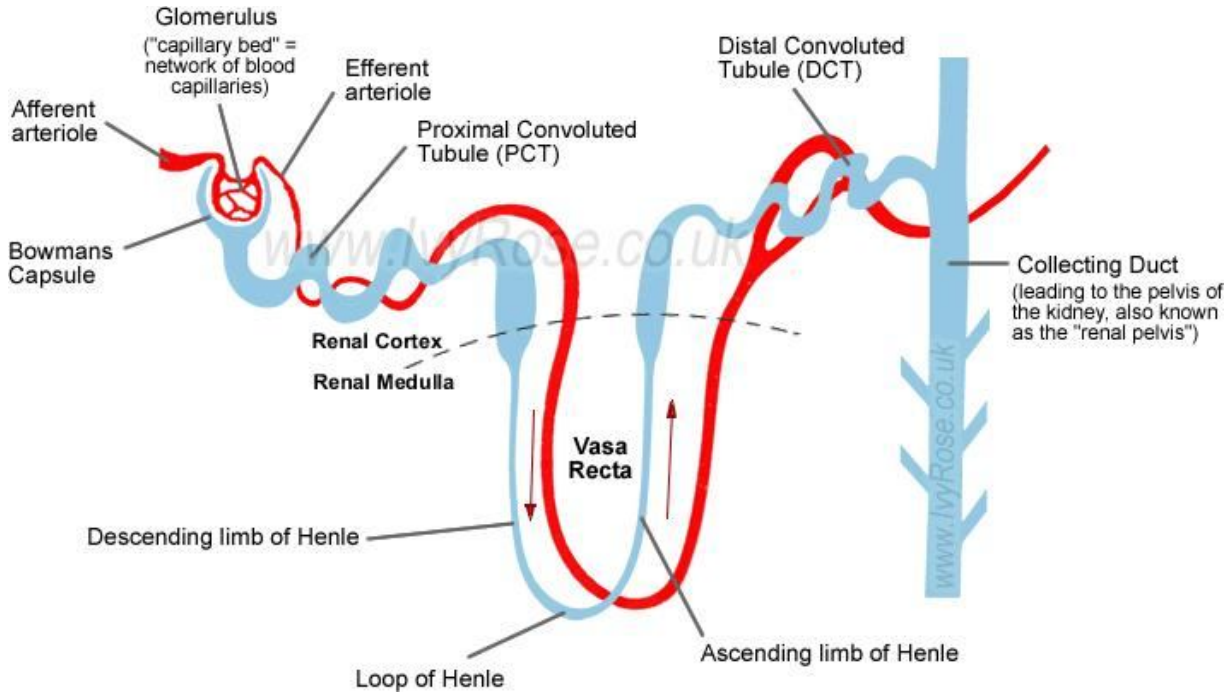
The weight of kidney approximately 120 g to 160 g in male. In female 110 g to 150 g . 10 cm to 12 cm in length, width is 5.0 cm to 7 cm in, and 2.0 cm to 3.0cm in thickness. sinus of the kidney consists of renal pelvis, the renal artery and vein, the lymphatics, and a nerve plexus . The organ is surrounded by a capsule. The capsule is smooth and easily removed. kidney is supplied by a single renal artery. In the hilar region renal artery divides into two branch. segmental arteries arise from the anterior branch and supply the upper, middle, and lower thirds of the anterior surface of the kidney .

The posterior branch supplies posterior surface. The apical segmental is a branch from the anterior division. There is no collateral circulation .

The aberrant arteries of the kidney arise from the from the superior mesenteric, testicular, or ovarian arteries. True accessory arteries supply the upper pole of the kidney.



THE NEPHRON



The nephron is the functional unit of the kidney . Each kidney contains about millions of nephrons. Each nephron has malpighian corpuscle , the proximal tubule, the thin limbs, the distal tubule, and the collecting tubule. The nephron orginate from the metanephric blastema. The connecting tubule derive from the metanephric blastema. The ureteric bud gives rise to collecting duct system says collecting tubule, the cortical coll ecting duct (CCD), outer medullary collecting duct , and the inner medullary collecting system. Functionally the nephron and the collecting duct system are acts in the same manner.

ACUTE KIDNEY INJURY :

DEFINATION: Acute kidney injury (AKI) is characterized by a rapid fall in the glomerular filtration rate . It also causes retention of nitrogenous waste products such as blood urea nitrogen (BUN) and creatinine[1].

The term acute renal failure constitutes of three phases . it includes initiation, maintenance, and recovery phases. each phases consists of variable duration and severity. On comparing to the chronic kidney disease the acute kidney injury is more dangerous one because even mild decrease in glomerular filtration rate may associated with unfavorable clinical outcomes.

AKI is classified asnonoliguric (urine output >500 mL/day), oliguric (urinary out-put <500 mL/day), or anuric (urinary output <150 mL/day)[2].Lower levels of urinaryoutput have consequence of severe initial injury, have implications for volume overload and electrolyte disturbances, and are of bad prognostic importance. However, the therapeutic depending onthe urine output does not change this prognostic association .

In view of diagnosis and management, AKI has been divided into three categories

1. Prerenal :

   Decresed blood supply to the one or both kidneys. Causes commonly hypotension of variable cause.

2. Intrarenal :

   Renal parenchymal tissue is involved.

3. Diseases involving large renal vessels,

PRE RENAL AZOTEMIA :

- Prerenal AKI is the most common cause of AKI and is an common physiologic response to renal hypoperfusion [3] and hypotension consequence, the special feature of this is , the integrity of renal parenchymal tissue is intact and GFR is corrected rapidly with restoration of renal perfusion.The clinical and biochemical features of prerenal ARF and ischemic ATN coexist in many patients.Severe renal hypoperfusion may cause ischemic ATN. Thus, prerenal AKI and ischemic ATN are part of a common manifestations of renal hypoperfusion,.

  Prerenal AKI can complicate any disease characterized by hypovolemia, low cardiac output, systemic vasodilatation, or intrarenal vasoconstriction True or "effective" hypovolemia leads to a fall in mean systemic arterial pressure, which, in turn, activates arterial and cardiac baroreceptors and initiates a series of neural and humoral responses that include activation of the sympathetic nervous system and renin-angiotensin-aldosterone system and release of antidiuretic hormone [4] . Noradrephrine, angiotensin II, and antidiuretic hormone act in concert in an attempt to maintain blood pressure and preserve cardiac and cerebral perfusion by stimulating vasoconstriction in relatively "less important" vascular beds such as the

musculocutaneousand splanchnic circulations, by inhibiting salt loss through sweat glands, by stimulating thirst and salt appetite, and by promoting renal water and salt retention.). Intrarenal biosynthesis of vasodilator prostaglandins (e.g., prostacyclin,), kallikrein and kinins, and possibly nitric oxide (NO) is enhanced. Angiotensin II may induce preferential constriction of efferent arterioles, probably because most angiotensin II receptors are found at this location Glomerular perfusion, ultrafiltration pressure, and filtration rate are preserved during mild hypoperfusion through several compensatory mechanisms. Stretch receptors in the walls of efferent arterioles detect a reduction in perfusion pressure, causing dilatation of afferent arteriolar smooth muscle cells and vasodilatation (autoregulation). As a result, intraglomerular pressure is m, the fraction is mintainedon of renal plasma that is filtered by glomeruli (filtration fraction) is increased, and GFR is maintained.

TABLE 29-2 -- Major Causes of Prerenal Azotemia

Gastrointestinal losses: vomiting, , diarrhea, nasogastric suction Hemorrhage: traumatic, surgical, postpartum blood loss,gastrointestinal, Renal losses: drug-induced or osmotic diuresis, diabetes insipidus, adrenal insufficiency. "Third-space" losses: pancreatitis, crush syndrome, hypoalbuminemia Skin and mucous membrane losses: burns, hyperthermia, and other causes of increased insensible losses
Comparomised cardiac output: Pulmonary hypertension, pulmonary embolism, positive-pressure mechanical ventilation

Diseases of myocardium, valves, pericardium, or conducting system Systemic vasodilatation Drugs: ACE inhibitors , afterload reduction drugs , anesthetics, drug overdoses Sepsis, liver failure, anaphylaxis
Renal vasoconstriction Noradrenaline, ergotamine, liver disorders ,, hypercalcemia, sepsis
Agents that acutely impair autoregulation and glomerular filtration rate in specific settings Severe renal hypoperfusion, Angiotensin-converting enzyme inhibitors in renal artery stenosis Inhibition of prostaglandin synthesis by NSAIDS during renal hypoperfusion

These compensatory renal responses are overwhelmed during states of moderate to severe hypoperfusion, and ARF ensues.). Intrarenal biosynthesis of vasodilator prostaglandins (e.g., prostacyclin, prostaglandin E_2), kallikrein and kinins, and possibly nitric oxide (NO) is enhanced. Angiotensin II may induce preferential constriction of efferent arterioles, probably because most angiotensin II receptors are found at this location of proximal tubules .Autoregulatory dilatation of afferent arterioles is maximal at a mean systemic arterial blood pressure of about 60 to 90 mm Hg, and hypotension below this level is associated with a precipitous decline in glomerular ultrafiltration pressure and GFR. Lesser degrees of hypotension may provoke prerenal AKI in the elderly, in patients with renovascular disease, and in patients with diseases affecting the integrity of afferent arterioles (e.g., hypertensive nephrosclerosis, diabetic nephropathy).In addition, very high levels of angiotensin II, as are found in patients with marked circulatory failure, provoke constriction of both afferent and efferent arterioles, thus negating the relatively selective effect of low levels of this peptide on efferent arteriolar resistance.

Nonsteroidal anti-inflammatory drugs (NSAIDs), including cyclooxygenase II inhibitors, decrease renal prostaglandin biosynthesis .Several classes of commonly used agents impair renal adaptive responses and can change compensated renal hypoperfusionto overt prerenal AKI or cause progression of prerenal AKI to ischemic ATN. Similarly, inhibitors of angiotensin-converting enzyme (ACE) and angiotensin II receptor blockers may trigger prerenal AKI in individuals in whom intraglomerular pressure and GFR are depends on angiotensin II .They do not compromise GFR in normal individuals but it may cause prerenal acute kidney injury in subjects with true hypovolemia or decreased effective arterial blood volume, or in patients with chronic renal insufficiency in whom GFR is maintained in part by prostaglandin-mediated hyperfiltration through remnant nephrons.. ACE inhibitors and ARBs attenuate these compensatory responses and can precipitate AKI in such patients. ACE inhibitors or ARBs, like NSAIDs, may also precipitate prerenal AKI [5] in patients with compensated renal hypoperfusion of other causes, needed close monitoring of the serum creatinine level when these drugs are administered to high-risk

individuals.

This complication is classically seen in patients with bilateral renal artery stenosis or unilateral stenosis in a normally functioning one kidney. Here, angiotensin II preserves glomerular filtration pressure distal to renal arterial stenosis by increasing systemic arterial pressure and by triggering selective constriction of efferent arterioles norepinephrine, angiotensin II, antidiuretic hormone. The classic urinary and biochemical sequelae of prerenal AKI can be predicted from the stimulatory actions of, and low urine flow rate on salt and water reabsorption from urine and include concentrated urine (specific gravity >1.016, osmolality >300mOsm/kg H_2O, low urinary Na^+concentration, and benign urine sediment.prerenal ARF may be seen in patients with renal concentrating defects in case of diabetes insipidus and in the setting of large endogenous or exogenous solute loads. Hypernatremia due to increased free water losses is a clue to the presence of a increase urinary output prerenal state.

Some vasoactive drugs, and diagnostic agents like iodide stimulate intense intrarenal vasoconstriction and it cause glomerular hypoperfusion and acute kidney injury with many of the functional, clinical, and biochemical features of prerenal AKI. Examples include hypercalcemia,

sepsis ,radiocontrast agents, calcineurin inhibitors (cyclosporin,tacrolimus), amphotericin B, cocaine, and noradrenaline (e.g., therapeutic administration, pheochromocytoma, brain damage). Tacrolimusand cyclosporine inhibitors precipitate ARF by inducing intrarenal vasoconstriction and hypoperfusion, and by stimulating mesangial cell contraction and a fall in glomerular filtration surface area. Tubular necrosis is rare in this setting, although long-term calcineurin inhibitiondrugs may lead to irreversible renal impairment, probably as a results of obstructive arteriopathy and chronic medullary ischemia producing necrosis .

Acute Tubule Necrosis :

The term ATN and the clinical term ARF/AKI are commonly used interchangeably when referring to ischemic and nephrotoxic renal injury but presence of frank necrosis of renal tubules and it may also sparse in most cases and vise versa. [6] , The duration of cardiopulmonary bypass and the degree of preoperative and postoperative complications leads to hypoperfusion and hypotension . this will aggrevate the acute kidney injury especially in older individuals like diabetic mellitus. It should also be noted that the that extracellular fluid losses in many clinical situations or transient renal hypoperfusion commonly in the settings of cardiac arrest or aortic cross clamping generally do not cause acute tubular necrosis in the absence of existing renal impairment or the presence of another nephrogenic insult says drug induced , sepsis, or rhabdoemyolysis, leukemaoid reactions, and other consequences . [1]

Prerenal AKI results from hypoperfusion. It will reverts once the reperfusion is maintained. But in case of intrinsic AKI it is associated with injury to renal tissue .It does not cured immediately on restoration of renal perfusion.

The term ATN and the clinical term ARF/AKI are commonly used interchangeably when referring to ischemic and nephrotoxic renal injury but presence of frank necrosis of renal tubules and it may also sparse in most cases and vise versa. Prerenal AKI and ischemic ATN are part of a two spectrum of manifestations of renal hypoperfusion or systemic hypotension . prerenal AKI being a response to mild or moderate hypoperfusion and ischemic ATN being the result of more, The duration of cardiopulmonary bypass and the degree of preoperative and postoperative cardiac severe or prolonged hypoperfusion often coexistent with other renal diseases especially in older individuals like diabetic mellitus is highly influencing . It should also be noted that the that extracellular fluid losses in many clinical situations or transient renal hypoperfusion commonly in the settings of cardiac arrest or aortic cross clamping generally do not cause acute tubular necrosis in the absence of existing renal impairment or the presence of another nephrogenic insult says drug induced , sepsis, or rhabdoemyolysis, leukemaoid reactions, and other consequences

Ischemic acute tubular necrosis occurs in the setting os prolonged addominal surgery,sepsis, dehydration and toxin mediated [7]. Acute tubular necrosis most commonlyoccurs in aortic surgery in case of undergoing emergency repair of ruptured abdominal aneurysms and other major intraabdominal surgery or after complicated elective procedures requiring prolonged (>45 minutes) clamping of the aorta above the origin of the renal arteries one one side . However, 50% of cases of postsurgical ATN occur in the absence of documented hypotension. ATN occurs in 50% to 80% of patients who suffer burns involving more than 25% of their surface area and it is a frequently multifactorial and due to the combined effects of hypovolemia, most commonly dehydration , rhabdomyolysis, sepsis, and nephrotoxic antibiotics. combination of systemic vasodilatation and intrarenal vasoconstriction is the mechanism behind the sepsis induces renal hypoperfusion provoking. [8]

Exotoxins and endotoxins results from variable causes can cause acute kidney injury. Nephrotoxic ATN occurs in pharmacologic agents and poisons. Some metabolic products also can cause acute kidney injury.It occurs in the settings when it present at high concentrations in

the circulation. This toxic products cause intrarenal vasoconstriction, direct tubule toxicity, and intratubular obstruction . In general, nephrotoxins cause renal The kidney is particularly vulnerable to nephrotoxic injuryby virtue of its rich blood supply. the countercurrent mechanisms of the medullary interstitium is responsible for this.

Postrenal Acute Kidney Injury

Urinary tract obstruction is one of the common cause for the acute kidney injury the obstruction can occurs anywhere between the urerter, renal pelvis, bladder, urethra, prostate disease, penile structures. prostatic disease ,. Neural tube defects can cause neurogenic bladder. Anti cholinergic drugs can also cause urinary tract obstruction. least commonly the Blood clotsin the urinary tract , calculi, and urethritis with spasm are less common causes of acute lower urinary tract obstruction .Ureteric obstruction may result from intra visceres obstruction like inadvertent surgical ligature. . This results in gradual distention of proximal ureter, renal pelvis, and other structures of neoprhons , and a fall in GFR. initial modest increase in renal blood flow, arterial vasoconstriction soon supervenes as a consequence of acute obstruction

leads to acute kidney injury.

PATHOGENESIS OF ACUTE KIDNEY INJURY :

The effect of renal injury, whether from ischemia or from other causes, is a decrease in the GFR. This decrease GFR often occurs in the absence preexixting evident damage to the kidney as seen on light microscopy. There are three major mechanisms are commonly responsible for the fall in GFR pertaining to acute kidney injury .The first mechanism pertaining to the drop in the filtration pressure in the glomerulus. This drop in pressure is reflected in the nephron in the formof caused by afferent arteriolar vasoconstriction and proximal tubular obstruction. This mechanism leads to a indirect fall in the GFR. Afferent arteriolar vasodilation is thought to be a result of endothelial cell injury caused by toxins and other insults This leads to an imbalance in vasoactive substances and counter current mechanism in the medullary interstitium with a predominance of vasoconstrictive activity. The second mechanism is tubular back-leakage syndrome characterized by the , leads to a fall in the GFR because of the leak in the tubules.. Damage and loss of epithelial cells denuded basement membranes and loss of tight junctions between those cells that are critical to maintaining separation of

tubular filtrate and the surrounding interstitium . of damage and loss of epithelial cells denuded basement membranes and loss of tight junctions between those cells that are critical to maintaining separation of tubular filtrate and the surrounding interstitium . This syndrome of Back-leakage of glomerular filtrate occurs in the setting Tight junctions are disrupted in the setting of adenosine triphosphate depletion, allowing back-leakage of potassium and other solutes into the renal interstitium. . THP tends tojoined and form a gel that can further trap cells and tubular cell debris following acute kidney injury ATN further promotes THP gel formation [9] The concentration of various molecules in the renal tubules in evolving. Besides a fall in GFR, there is also a decreased ability of the kidney to concentrate urine following AKI.

Mediators of Inflammation :

Inflammation plays a central role in AKI. Repair, inflammatory cells and soluble mediators are likely major determinants of the outcome from ARF. [10]From initiation to extension through A number of different inflammatory cells and soluble chemokines have been found to be thought for full renal damage and loss of glomerular filtration to occur. Inflammatory pathways are attractive targets for therapy, and there has

been great success with interventions in experimental models of AKI. blocking inflammation after the renal insult has occurred affords much less protection to the kidney is the limitation of this approach seems to be that

Inflammation in microvessels :

The microvasculature injury is the foremost events leading to damage of renal tubular epithelial cells. The kidney receives 25% to 35% of cardiac output, and most of that blood flow is directed to the renal cortical structures.[11] The vasa recta branching from efferent arterioles, , eventually become the vessels of Postglomerular vessels. countercurrent multiplier, allowing for more trafficking of water and solutes is the low-flow state in the vasa recta is a critical aspect of the. However, the low-flow state leaves the medulla more commonly hypoxic when compared with other regions of the renal structures . Very slight decreases in the blood flow and oxygen delivery can lead to hypoxic damage. The reason behind the renal cortex with a partial pressure of oxygen of about 60 mm Hg, the medulla has a partial pressure of oxygen in the 20 to 30 mm Hg range . so consequently, Anoxic injury to local cells, including vascular smooth muscle cells resultant disruption of their cytoskeleton.This

cellular deformities erythrocyte interactions and promotes sludging of erythrocytes in a and hypoxia in and around the microvasculature leads to endothelial- way that is analogous to a sickle cell vaso-occlusive crisis.12. Vascular congestion with RBC sludging has been described on renal biopsies. These studies have demonstrated that structures around the vase recta capillaries had stopping of of blood flow following an ischemic event as compared with intraglomerular capillaries that had diminished but never absent flow. Red blood cells Peritubular capillaries also took longer to recover normal blood flowin comparison with other intrarenal vessels. The combination ofanoxic injury, changes in endothelial cell morphology, contributes to our understanding of the regionalization of injury within the kidney

and heightened interactions between and endothelium leads to the extension of the initial renal injury .

Leukocytes as mediator :

On biobsies of kidney neutrophils Infiltration are infrequently seen of human ATN but it was known that infiltrate the kidney following an ischemic insult in experimental animals . Decreased renal injury occurs

when neutrophil migration and activity are blocked in some experimental studies have demonstrated[13]. Early inflammation is classically supported bymargination of neutrophils to endothelium.

Integrin pathway modulation can alter outcome of renal failure through neutrophil-independent ways . selectin ligands to reduce ischemic kidney injury in deceased donor transplants is confirmed multicenter clinical trial is under way blocking.

Blockade of the integrin CD11/CD18 protects from ARF .

There are many other key chemokines pathways that mediate the pathogenesis of AKI. Toll receptors also found to have likely play an important role. TLR1 has been shown to mediate experimental ischemic ARF, and TLR43mediates ARF in a mouse lipopolycechride model. . Many of the cytokines play a role in ARF, which is a rapidly advancing and interesting area of investigation TLR8 and MyD88 have been implicated in a mouse sepsis model.

Apoptosis

Apoptosis pathophysiologically related to acute renal failure . cellular necrosis and apoptosis are different . swelling of cells, loss of plasma

membrane integrity, by and eventually cell rupture with spillage of cellular constituents cellular necrosis. is characterized contents into the extracellular space[14]. In apoptosis, the cell nucleus and cytoplasm condensation and then split into smaller small bodies. Also note that cytoplasmic organelles, including the mitochondria, are are phagocytized by macrophages or other cells, to cause inflammation.

The effects of apoptosis on the host can change during the course of AKI, ranging from deleterious to whereas during the convalescent phase, apoptosis may be an important mechanism to regulate cell number and structure . beneficial, depending on the phase of acute kidney injury . Initially, apoptosis may be deleterious to the kidney and overall renal function, The signs of apoptosis in the kidney, within 10minutes of a anoxic insult in the rat kidney. 15 These early findings of apoptosis removal of necrotic tubular cells from the area, and may be a way to often precede any can cause deterioration in renal initially heralded by DNA fragmentation in the cells of the thick ascending limb, can be seen as a function. A second peak in the amount of apoptosis in renal tissue occurs days to weeks after the initial insult. This often follows help regulate the number of newly generated cells.

. Studies of cell culture found that the duration and severity of that injury often correlates with the type of donor, deceased or living, and the cold ischemic time. the initial hypoxic injury triggers apoptotic pathways in some cells, but Ischemic renal injury can occur in donar kidney in renal transplantation patient. if there were differences in rates of apoptosis diseased donor kidneys were compared with living donor kidneys to determine. Very little apoptosis was found in the kidneys of live donors. Apoptosis was seen in all deceased donor patients , with a differences between the duration of cold ischemic time and the amount of cell death .

Animals treated with the caspase inhibitor before renal IRI had significant protection from renal damage and loss of renal function, but that protection was lost if animals were treated following the onset of apoptosis. The expression of caspases in the cascade of events leading to apoptosis was found to be a critical step in the release of proinflammatory mediators, such as endothelial monocyte-activating polypeptide-II, which can further induce P- and E-selectins.

Blocking caspase activity and apoptosis has been the target of other interventions, often successfully limiting renal damage in models of ischemic injury. Minocycline, the tetracycline antibiotic, was shown to

block renal damage through reduction of apoptosis in a rat model of IRI. Other studies have targeted different pathways of apoptosis EPO administration can protect the kidney from the effects of IRI in experimental models [16]. Many investigators have shown that EPO can decrease the number of apoptotic tubular epithelial cells from an otherwise lethal dose of cisplatin. effectively blocked caspase cells following an ischemic insult. Apoptosis has been shown to be an important pathway of injury in the kidney in other models as well. The use of cell cycle inhibitors that also -3 were found to protect cultured renal Cisplatin is a highly nephrotoxic chemotherapeutic agent that damages cells .

The Endothelial Cell

When an initial insult damages the endothelium of the renal vessels, the result is an endothelial bed that is ineffective in regulating local blood flow and cell migration into tissues, and preventing coagulation. Damage to endothelial cells can also favourAKI ..This vascular dysfunction leads to continued ischemic injury following any insult, the extension phase of AKI. The structural alterations that occur in the endothelial cell following an ischemic injury have been partially elucidated and help explain the

functional changes that occur during this injury process. The baseline structure of the endothelial cell is maintained by a network of protein filaments that make up the cytoskeleton. Actin filament bundles, which have been shown to shrink in the setting of ATP depletion. The assembly actin desaturasing factor/cofilin (ADF.) family of proteins is known to regulate actin dynamics and play a role in the changes to the actin cytoskeleton during ischemia under ATP-depleted conditions (such as ischemia). Modulation of the ADF/cofilne mediated ADF/cofiline has a concentration-dependent effect on changes to the actin cytoskeleton changes to the actin cytoskeleton in ischemic endothelial cells has potentially important therapeutic implications for ischemic AKI [17,] and may have applications in other organ systems as well.

The Renal Tubular Epithelial Cell

Following are the most obvious cell type injured in ARF that is the renal tubular epithelial cells, which are visible on routine urine analysis and light microscopy as well as. Injury and loss of epithelial cells, through necrosis or apoptosis can lead to loss of kidney function and apparent drop in GFR through processes of glomerular filtrate and tubular obstruction. The renal tubular cell has a marked ability to

recover from an ischemic injury. The tubular epithelial cell progresses through a series of structural changes that finally leads to restoration of normal structure tubular lumen, and the remaining viable cells dedifferentiate and proliferate leading to final reformation of normal epithelium. The initial and function. These steps include an initial loss of cell polarity and brush border, which contributes to altered solute tracking Some cells die and are sloughed into the insult to the tubular epithelial cell This structural change in the cell leads to the formation of membrane dependent depletes cellular ATP, which, in turn, leads to disruption of the basal actin cytoskeleton in a fashion that mirrors the changes in vascular endothelial cells in thekidney. vesicles or blebs that can either be secreated into the tubular lumen as part of the cellular debris leading to cast formation and tubular obstruction

COURSE OF ACUTE TUBULE NECROSIS

Initiation, maintenance phase, and recovery phases are the three phases of ATN .[18] The initiation phase is the period when patients are exposed to the ischemia or toxins and parenchymal renal injury is evolving but not yet established. Acute tubular necrosis is potentially preventable during

this period, which may last hours to days. The maintenance phase is the most important phase in the prognosis of acute kidney injury . avoidance of toxin and the toxic products reperfusion , all had a important role in the outcome of acute kidney injury . interventions in the form of drugs says asmotic diueretics, loop diuretics, control of diabetic mellitus and hypertension all influence the recovery phase. In case of recovery phase it is assessed by adeguate urinary output, decrease in urea ,creatinine, symptomwise improvement . Occasionally, diuresis may be inappropriate and excessive if recovery of tubule reabsorptive processes lags behind glomerular filtration.

COMPLICATIONS OF ACUTE KIDNEY INJURY

Intravascular volume overload is an almost inevitable consequence of diminished salt and water excretion in AKI and may present clinically as mild hypertension, increased jugular venous pressure, bibasilar lung crackles, pleural effusions or ascites, peripheral edema, increased body weight, and life-threatening pulmonary edema. Moderate or severe hypertension is unusual in ATN and should suggest other diagnoses Hypervolemia may be particularly troublesome in patients receiving multiple intravenous medications, sodium bicarbonate for correction of

acidosis, or enteral or parenteral nutrition. such as hypertensive nephrosclerosis, glomerulonephritis, renal artery stenosis, and other diseases of the renal vasculature. [19]Excessive water ingestion or administration of hypotonic saline or isotonic dextrose solutions can trigger hyponatremia, which, if severe, may cause cerebral edema, seizures, and other neurologic abnormalities.[20]

agnesium, magnesium-containing laxatives, or antacids). Hypomagnesemia occasionally complicates nonoliguric ATN associated with cisplatin or amphotericin B and, as with hypokalemia, probably reflects injury to the thick ascending limb of loop of Henle, the principal site for Mgreabsorption. Hypomagnesemia is usually asymptomatic but may occasionally be manifest as neuromuscular instability, cramps, seizures, cardiac arrhythmias, or resistant hypokalemia or hypocalcemia.

Mild gastrointestinal bleeding is common (10% to 30%) and is usually due to stress ulceration of gastric or small intestinal mucosa.Alterations in neurologic function may reflect the onset of the uremic syndrome, metabolic complications of AKI, impaired excretion of prescribed neuropsychiatric medications, or primary neurologic disease including TTP.

.

MANAGEMENT OF ACUTE KIDNEY INJURY

The goals of management of AKI encompass the need to prevent death, ameliorate metabolic and extracellular volume complications, and preserve renal function so as to prevent the development of chronic kidney disease.

Prerenal Acute Kidney Injury

Prerenal AKI is reversible on restoration of renal perfusion. The replacement fluids varies depending on the source of fluid loss for treatment of hypovolemia. packed RBCs is used for hypovolemia caused by hemorrhage .it is used only if the if the patient is hemodynamically unstable or if the hematocrit is low. isotonic saline alone can be used in the absence of active bleeding or hemodynamic instability.. Recent critical reviews of RCTs comparing crystalloid with colloid replacement for resuscitation in critically ill patients conclude that the routine use of colloids may be associated with an adverse outcome and is not justified. [21] Colloid solutions should be used only sparingly, with regular monitoring

of renal function and the risk of hyperoncotic renal failure minimized by the concomitant use of appropriate crystalloid solutions. In case of loss due to urinary or gastrointestinal loss vary hypotonic fluids can be used. in this case initial replacement is best achieved with hypotonic solutions (e.g., 0.45% saline), and subsequent fluids is depends on composition of excreted or drained fluids. Electrolytes should be monitored in all subjects. K^+ supplementation is required in case of hypokalemia occurring during treatment of metabolic acidosis

Intrinsic Acute Kidney Injury

Optimization of cardiovascular function and intravascular volume is the single most important maneuver in the management of intrinsic AKI. Aggressive management of fluid loss in case associated with major trauma can prevent the major consequenceof intrinsic acute kidney injury .22.Sepsis related AKI is a common clinical presentation and is associated with mortality rates as high as 80%. Volume depletion has been identified as a risk factor for nephrotoxic ATN induced by radiocontrast material.[1] Restoration of volume prevents the development of experimental and human ATN in many of these settings. The importance of maintaining euvolemia in high-risk clinical situations has been

demonstrated most convincingly with contrast nephropathy, in which close attention to intravascular volume status ensures a low frequency of AKI.

Some drugs like loop Diuretics or other class of diuretics should be used in caution . simillarly drugs like NSAIDs , ACE inhibitors, and other vasodilators should be used with caution., because they may convert prerenal ARF to ischemic ATN and sensitize such patients to the actions of nephrotoxins. Careful monitoring of circulating drug levels appears to reduce the incidence of ARF associated with aminoglycoside antibiotics or calcineurin inhibitors Interestingly, the antimicrobial efficacy of aminoglycosides appears to persist in tissues even after the drug has been cleared from the circulation. Also, there is convincing evidence that once-daily dosing with these agents affords equal antimicrobial activity and less nephrotoxicity than conventional regimens. [23]The use of lipid-encapsulated formulations of amphotericin B may offer some protection against renal injury. Several other agents are commonly employed to prevent AKI in specific clinical settings. The drug called Allopurinol is useful for acute urate nephropathy; but many occasional patients receiving allopurinol still develop AKI, probably by toxic actions of

hypoxanthine crystals on tubule function.[24].so nowadays commonly the use of recombinant urate oxidase called raburicase at a dose of 0.05–0.2 mg/kg should be considered. Lets see how it acts Raburicase promotes the degradation of uric acid to allantoin . so it proven efficacy both as prophylaxis and treatment for acute uric acid-mediated tumor lysis syndrome. Also note that [1] In oligoanuric patients, prophylactic hemodialysis to remove excess uric acid is important.

A newer drug called Amifostine, known to be an an organic thiophosphate, has been used to to ameliorate cisplatin nephrotoxicity in patients with solid organ or hematologic malignancie. a good droug named dimercaprol, a chelating agent, may prevent heavy metal nephrotoxicity. Ethanol by dumping the effects of ethylene glycol metabolism to oxalic acid and other toxic metabolites but has been superceded by the introduction of fomepizole, known fo its active effective alcohol dehydrogenase inhibitor that decreases production of ethylene glycol metabolites and therefore prevents the development of renal injury.

Specific Therapies

For the past 2 decade there has been extensive investigation into the

pathogenesis of AKI using experimental animal models and also commonly by the cultured cells. These studies have led to substantial advances in our understanding of the mechanisms that could potentially play a role iof some drugs in ATN in humans. This information has led to an exciting array of potentially difficult targets for the treatment of this common and serious disease. However, a number of interventions shown to be effective in treating AKI in animals have failed to be effective in humans with ATN. There are so many possible reasons for lack of success in translating therapeutic successes for AKI from out of scene .We lack adequate information regarding the pathology of ATN in humans in the current era, because there has been a lack of systematic studies in this area for many years. It is possible that human tissue, subjected to conventional histologic stains would facilitate the identification of those patients most likely to response to treatment.

Dopamine

(1 to 3 mg/kg/min) is the renal doseof dopamine has been widely advocated for the management of oliguric AKI. [24]Note that in experimental animals and healthy human volunteers, renal dose dopamine increases renal blood flow and, albeit to a lesser extent, GFR. It was well

estabilished that r enal dose dopamine has not been demonstrated to prevent or alter the course of ischemic or nephrotoxic ATN in controlled clinical trials. [25]But what to do the available evidence would suggest lack of efficacy. Also note that dopamine, even at low doses, is potentially toxic in critically ill patients and can induce tachyarrhythmias, myocardial ischemia, hypotension extravasation necrosis among other complications. From this it was clear that the routine administration of dopamine to patients with oliguric AKI is not justified based on the balance of experimental and clinical evidence.

Fenoldopam

Fenoldopam is a new drug commonly used nowadays as a selective postsynaptic dopamine agonist mainly of D1-receptorsthat mediates more potent renal vasodilatation and natriuresis when compare to dopamine[1] However, it also promotes hypotension by decreasing peripheral vasculature resistance. In clinical trails early results suggest that positive results from small studies suggested a possible benefit renoprotective effect of fenoldopam in many clinical situation. But u should also note that , a subsequent larger randomized trial comparing fenoldopam to standard hydration in patients of angiographic procedures

found no benefit. Finally in a large RCT, fenoldopam administration did not reduce mortality or the need for renal replacement therapy in ICU patients presenting with early ATN.

Natriuretic Peptides

Coming to the ANP it is a 28-amino acid polypeptide synthesized in cardiac atrial muscle. ANP augments GFR by triggering afferent arteriolar vasodilatation and increasing GFR . In addition, ANP also inhibits sodium transport and lowers carbondioxide and bicarbonate requirements in several nephron segments. Synthetic preparation of ANP have shown promise in the management of ATN in the presence of laboratory setting. To date, this promise has failed to translate into clinically inadvesible benefit and also note that large multicenter, prospective, randomized placebo controlled trial of a drug called anaritide, a synthetic analog of ANP, failed to show clinically significant improvement in dialysis-free survival or overall mortality in ATN.[615] Subgroup analysis is failed to suggested an improvement in dialysis-free survival in treated patients, but this was not confirmed in a subsequent prospective trial of patients with oliguric AKI. a non marketable drug

Ularitide is a natriuretic pro-ANP fragment produced within the kidney. In a small randomized trial, it is also known that ularitide did not reduce the need for dialysis in patients with AKI.

Loop Diuretics

Using of high-dose intravenous diuretics to individuals with oliguric AKI is commonly practiced nowadays everywhere. 617] Although this strategy may minimize fluid overload,but there is no evidence that it alters mortality or dialysis-free survivalin majority of patients .it was also shown that Some retrospective analyses have reported an increased risk of death in subgroup of patients and poor recovery of renal function in patients treated in this manne. In case of a recent large RCT, high-dose intravenous loop diuretics augmented urine output but did not change the outcome of established AKI.[619] Given the risks of loop diuretics in AKI, including irreversible ototoxicity and exacerbation of ofexixtingAKI,so it should be keep in mind that their use should be restricted to the conservative management of volume overload .

Mannitol

There is no enough trails to support the routine administration of mannitol to oliguric patients. However it was known that when administered to severely oliguric or anuricpatients,it may trigger expansion of intravascular volume and pulmonary edema but severe hyponatremia owing to shift of water from the intracellular to the intravascular space is a common problem .[26]

In case of Glomerulonephritis or vasculitis corticosteroids can be used . in some rare situations caused by other intrinsic renal diseases such as acute it may also shown that it may respond to alkylating agents, and plasmapheresis, Corticosteroids appear to fasten remission in some cases of interstitial nephritis. Also known that Plasma exchange is useful in treatment of TTP and possibly sporadic hemolytic uren=mic syndrome in adults. The role of plasmapheresis in the drug-induced microangiopathies is not clear, and removal of the agent is the most important initial therapeutic method. HUS in children secondary to diarrhoea is should be managed conservatively and supporting evidence exists suggesting that early antibiotic therapy may actually promote the development of HUS in such patients. many RCP studies found that plasmapheresis may be of

benefit in ARF due to cast nephropathy in case of myeloma. Clearance of circulating light chains with concomitant drugs like hydroxyurea to decrease the rate of production had been postulated to reverse renal injury in patients with circulating light chains, multiple myeloma and AKI. A recent largeRCT compared plasma exchange and chemotherapy with chemotherapy alone. The study did not demonstrate improvement with plasma exchange when comparing with the death ratio , dialysis dependence, or GFR less than 30 mL/min at 6 months, and its routine use in this setting can no longer valuable.

Controlling of hypertension is very very important in certain situations like preeclampsia, malignant hypertension, and hypertension in case of chemotheraphy indused like cyclosporine .

Management of Complications

Metabolic complications such as hyperkalemia, hyperphosphatemia, and metabolic acidosis, intravascular volume overload, are almost occur in oliguric AKI, and preventive measures must be taken from the time of diagnosis.Prescription of nutrition should be offered to meet caloric requirements and minimize catabolism. In addition, doses of drugs excreted through the kidney must be adjusted according degree of renal

diseases.

After correction of intravascular volume deficits, salt intake should be adjusted to match losses such as urinary, gastrointestinal, drainage sites, insensible losses.By restriction of salt and water intake and by use of furosemide intravascular volume overload can usually be managed . However no proven rationale for routine administration of furosemide with AKI other than to treat this complication. In the volume-overloaded patient, high doses of loop diuretics or sequential thiazide and loop diuretic may be required if they fail to respond to conventional doses. Diuretic therapy should be discontinued in resistant patients to avoid complications presenting with ototoxicity. Caution should be exerted in the use of drugs that require an obligate sodium and fluid load. dialysis may be required for removal of volume when conservative measures fail. Hyponatremia are corrected by restriction of water intake and hypernatremia are corrected by treated by administration of water, hypotonic saline solutions, or hypotonic dextrose fluids.

Hyperkalemia of mild severity says <5.5 mEq/L) should be managed by restriction of dietary potassium intake and elimination of potassium rich

foods and potassium-sparing drugs . Moderate hyperkalemia said to be (5.5 to 6.5 mEq/L) in patients without clinical or ecg evidence of hyperkalemia can usually be controlled by administration of K+-binding ion exchange resins commonly used sodium polystyrene sulfonate at a dose of 15 to 30 g every 3 or 4 hours with sorbitol at a dose of 50 to 100 mL of 20% solution by mouth or rarely as a retention enema. Furosemide increase K+ excretion in diuretic-responsive patients. Emergency treatment is required when serum K+ values greater than 6.5 mEq/L and also in patients with ecg abnormalities or clinical features of hyperkalemia. In this case Intravenous insulin glucose solutions promote K^+ shift into cells in 30 minutes, a benefit that lasts for several hours. Sodium bicarbonate also promotes rapid onset less than 15 minutes, duration 1 to 2 hours shift of K+ into the intracellular space as does done by nebulized (5–10 mg) albuterol. Sodium polysterenesulfonate and sodium bicarbonate have sodium load; these compounds should be used carefully foroliguric patients to avoid intravascular volume overload and in case of pulmonary edema. calcium gluconate (10 mL of 10% solution intravenously over 10 minutes antagonize the cardiac and neuromuscular effects of hyperkalemia and is a emergency temporizing measure in case of whereas other agents reduce

serum K^+ concentration. Hemo or peritoneal d ialysis is indicated if hyperkalemia is resistant to all those measures.

Metabolic acidosis usually not require treatment unless the serum HCO_3 concentration falls below 15 mEq/L. severe acidosis can be corrected by either oral or intravenous bicarbonate administration. Initial rates of replacement should be depends on the estimates of HCO_{3^-} deficit and adjusted thereafter based on to serum levels. Patients should be watch for complications of bicarbonate including metabolic alkalosis, electrolyte imbalance and volume overload, and pulmonary edema. of oral administration of phosphate Hyperphosphatemia can usually be controlled by restriction intake and administration of agents such as aluminum hydroxide, calcium carbonate that reduce absorption of $PO4^{3-}$ from the gastrointestinal tract and also by oral administration of phosphate Hypocalcemia does not usually require treatment until it is severe, as may occur in patients with rhabdomyolysis or after administration of bicarbonate. Hyperuricemia is usually not severe in ARF and does not require specific intervention.

Physicians, nurses, and dietitians play a role in nutritional management in patients with AKI requires close collaboration among. Patients with

ARF represent a dispersed group and individualized nutritional management is required, especially in critically ill patients on renal replacement therapy in whom protein catabolism is more.27 . Diet is very important in case of management of acute kidney injury. Because the loss of nitrogenous products can cause decrease in lean body mass and subsequent malnitrtion and its consequences. If the duration of renal insufficiency is likely to be small and the patient is not much catabolic, then dietary protein should be restricted t is less. Catabolic patients, in whom on continuous renal replacement therapy, may receive up to 1.4 mg/kg body weight/day. Also the Total caloric intake should not exceed 35kcal/kg body weight/day . The enteral route of nutrition is preferred, because it prevents the morbidity associated with parenteral nutrition while providing support to intestinal function. Management of nutrition is much easy in nonoliguric patients and after commencement of dialysis.. Water-soluble vitamin supplementation is advised with the exception of some , which can, promote urinary oxalate excretion and stone formation.

Anemia may necessitate blood transfusion or administration of recombinant human erythropoietin if severe . Uremic bleeding rarely usually responds to desmopressin, correction of anemia, estrogens, or

dialysis. Renal Dosage adjustment is needed of drugs that are excreted by the kidney. [1] Gastric stress ulcer prophylaxis is commonly not indicated unless the patient is intubated. Febrile patients must be investigated aggressively for infection and may require treatment with broad-spectrum antibiotics while awaiting results of specific organisms. Meticulous care of intravenous needles , Foley catheters, and other devices is mandatory. It is not shown that prophylactic antibiotics have not been shown to reduce the incidence of infection in these high-risk patients.

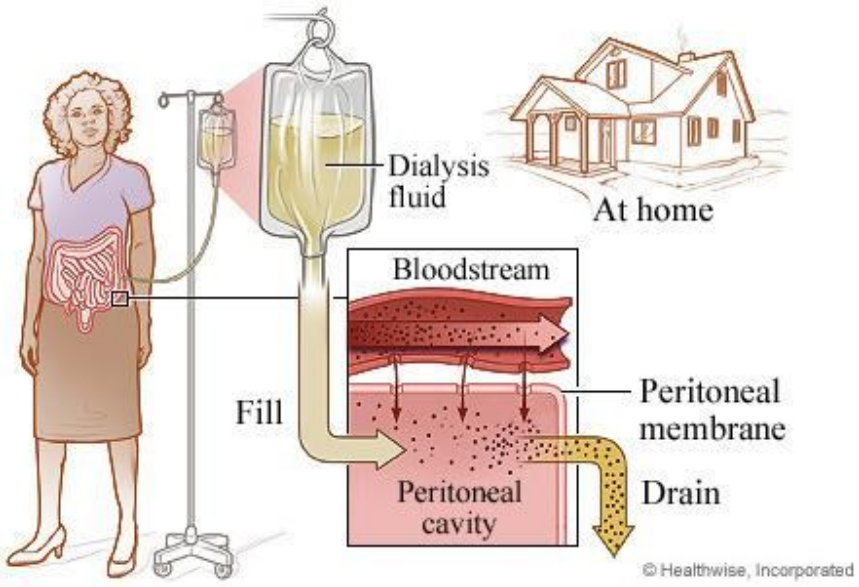
Indications and Modalities of Dialysis

General Comments

Dialysis does not fasten recovery from AKI.[28] but Initial studies suggesting that early dialysis therapy improved prognosis for patients with AKI have but it not proved not been confirmed. Similarly, there is no results on the optimal renal replacement therapy in AKI. The preferred mode of renal replacement therapy is an area of still research one. The claimed superiority of the continuous renal replacement techniques also remains unproven. Neither are there guidelines on the initiation of dialysis in AKI. following are the Absolute indications for the commencement of renal replacement therapy say symptomatic

uremia like asterixis, pericardial rub, encephalopathy and acidosis, hyperkalemia, or volume overload that proves refractory to medical management. However, in clinical practice, most physician initiate renal replacement therapy (RRT) before the onset of overt metabolic disorders when the need for renal support appears is not there . The choice of dialysis modality is often guided by the resources of the health care institution, the technic opinion of the physician and the clinical status of the patient.

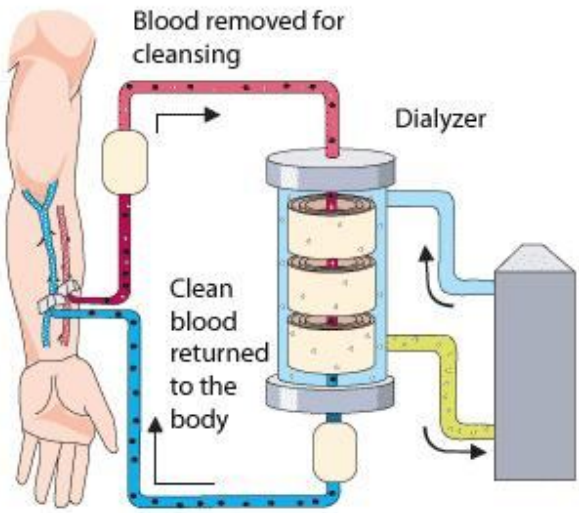
Peritoneal Dialysis



Peritoneal dialysis in AKI is done through a temporary intraperitoneal catheter. With the development of the slow continuous blood

purification therapies, there is a decline in the use of peritoneal dialysis in the acute setting.[29] It is still used in the treatment of AKI in regions where access to acute intermittent or slow continuous hemodialysis is very low. Peritoneal dialysis has the advantage of being relatively portable, thus facilitating its use in remote or resource- timed areas. Systemic hypotension is typically avoided, and other benefits include the lack of systemic anticoagulation and need for angioaccess as soon As possible Solute clearance and control of metabolic disturbance in critically ill patients may be inferior to continuous veno-veno hemofiltration, and this has been associated with an adverse outcome inAKI secondary to infection. Other drawbacks include the risk of visceral injury during catheter placement and peritonitis can occur .

Acute Intermittent Hemodialysis



Before the invent of hemodialysis, renal replacement therapy was commonly done. It was done in alternate days for 2 to 3 hours each time. Here double lumen catheter is passed through either jugular vein or femoral vein . Subclaviancanulation offers an alternative but it causes high rates of venous stenosis and is best avoided.[30] To note is the Femoral vein catheterization is technically easy and relatively free of complications. It is useful in patients who cannot tolerate the left lateral position or who require only a short treatment course (e.g., removal of an exogenous toxin). Jugular lines are preferred for more prolonged treatment courses, but with careful nursing management, it is possible to maintain a femoral line in the bedbound patient without causing a significant excess infection risk. The choice of membrane used during

dialysis also have an effect on outcome. Several, although not all, RCTs indicate that the maintenance phase of ATN is significantly shorter by using synthetic dialysis membranes (e.g., polysulphone, polyacrylonitrile) than with other membranes. However, systematic reviews of the literature have failed to demonstrate a benefit of synthetic over more modern substituted membranes.

During acute intermittent dialysis Anticoagulation with heparin must be used for preventing thrombosis of the extracorporeal circuit. Routine bedside assesement of the activated clotting time (ACT) allows heparin dosage adjustment as required to maintain a normal baseline value. Also note that Heparin-free dialysis can be performed in patients with hemorrhagic complications. This involves coding the dialyzer with a heparinized solution (3000U/L) and setting the blood flow rate at least 200 to 250 mL/min. A periodic saline rinse also should be used which should be done every 30 minutes to prevent the clotting in the extracorporeal circuit. If heparin-induced thrombocytopenia (HIT) is a concern then the heparin coating should be avoided. Other anticoagulation techniques include the usage of a single bolus of low-molecular-weight heparin at the start of dialysis.Less used anticoagulant

strategies include regionalheparinization with protamine administration in the venous return line, regional citrate , continuous prostacyclin infusion, and use of direct thrombin inhibitors: says hirudin, lepirudin .

. Intradialytic hypotension is common in patients undergoing this type of hemodialysis. Hypotension impairs solute clearance and the efficiency of dialysis. In addition it can further compromise renal perfusion and exacerbate tubular necrosis . Intradialytic hypotension is typically caused by excessive fluid removal during ultrafiltration. The latter, in turn, may occur if there is volume overload if the fluid removed is not matched by flux of fluid into the intravascular space from interstitial and other compartments, if the volume of fluid removed is more , or if the patient's compensatory responses are less as a result of microvascular disease or vasodilatatory medications say nitrates, and antihypertensive medication. Hypotension is a problematic in critically ill patients with ATN and concurrent sepsis, hypoalbuminemia, malnutrition, or large third-space fluid losses. Management of intradialytic hypotension requires careful assessment of intravascular volume, by invasive intra catheter monitoring, if necessary; usage of realistic ultrafiltration targets; and close observation for tachycardia or hypotension during dialysis. The

immediate management of hypotension involves the discontinuation of dialysis , placing the patient in the Trendelenburg position, and the rapid infusion of normal saline.

The dialysis disequilibrium syndrome is acommon complication or side effects of hemodialysis . it is because of cerebral s[edema. The cause of cerebral edema is not known . however it was hypothesized that sudden removal of solutes can cause the protective substances in the brain to be removed and cause cerebral edema as a consequence.[31] It can be slowly overcome with the application of slow rate hemodialysis in the starting period. The precise prescription of dialysis to achieve this outcome depends on such variables as membrane size, blood flow rate, and duration of treatment. Typically, this will involve an initial treatment time of approximately3 hours with a blood flow rate of 300 to 350 mL/min. Isolated ultrafiltration can continue for a longer period if volume removal is the critical management issue.

Once the patient is established on dialysis, the optimal dose of dialysis is depends on many factors . The standard protocol for dialysis adequacy using intermittent hemodialysis in ARF is not defined. Of note, the bad nutrition state observed in ill patients may justify large dialysis dose . In

patients with AKI, the change between delivered versus prescribed hemfiltration dialysis dose may be significant change to hemodynamic instability like filter clotting and inadequate vascular access, among other reasons. A randomized trial of daily versus alternate-day hemodialysis suggested a significant mortality in patients receiving daily dialysis. According to this study daily hemodialysis offer better uremic control while facilitating more intensive catabolic support without additional hemodynamic compromise and is now considered the standard of care.

The potential importance of dialysis membrane as a determinant of outcome in ATN has been discussed before . Occasionally robust complement and leukocyte migration by cellulosic membranes is followed by leukocyte attachment in the lungs, hypoxemia, dyspnea, and back pain; this is also termed the the first use syndrome. More indolent anaphylactoid reactions were seen in the past as a result of hypersensitivity to ethylene used to sterilize the dialysis circuit. These reactions are now not common as a result of changes in commercial sterilization techniques and the routine washing of the dialysis circuit. Patients who receive concomitant ACE inhibition can rarely have anaphylactoid reactions are observed in patients dialyzed on AN60

synthetic membranes . AN69 activates the kallikrein system and promotes bradykinin synthesis . as we all know the breakdown of bradykinin is inhibited by ACE inhibition so that in combination, ACE inhibitors and an AN69 membrane accentuate the circulating bradykinin levels.

Continuous Renal Replacement Therapy

As we know most patients in acute tubular necrosis are ill, hypercatabolic, and hemodynamically unstable. They frequently have large obligate fluid requirements, although on intravenous medication and parenteral supplements . In this condition , filtration of large volumes of plasma over a relatively short period by acute hemodialysis may induce circulatory compromise. Even if tolerated , acute intermittent hemodialysis may not achieve adequate filtration or solute clearance to avoid pulmonary edema and uremia, and continuous renal replacement therapy (CRRT) may be more reasonable . Hemofiltration was first described in 1979 for the management of refractory edema. For the past 3 decades have yielded a variety of slow continuous dialytic therapies. Whereas the various techniques differ in their technical detail, they share several features such as relative simplicity of operation, the ability to remove large volumes of fluid with minimal hemodynamic compromise,

and the capacity to control uremia and electrolyte and acid-base abnormalities with minimal change of plasma osmolality.

OUTCOME

The mortality rate approximates 50% among patients with intrinsic AKI. It was changed in the last two decades because of active management protocols This lack of improvement in outcome, may be more apparent than real and reflect a reduction in the percentage of AKI in addition with an with an increase in AKI complicating the multiple-organ dysfunction. When combined with the current trend for more aggressive surgical and intervention in the aged population, these factors probably mask an improvement . Mortality rates differ markedly depending on the cause of AKI: approximately 10% in obstetric patients, 20% in toxin-related AKI, and 50% to 80% in patients with sepsis. Although it was once held that the provision of effective renal replacement strategy largely change the prognostic import of an episode of AKI, more recent observations clearly demonstrate that this is not case . and that all too often, the development of AKI directly contributes to poor patient outcomes.Following are factors causing poor prognosis include male sex, advanced age, oliguria , and a rise in the serum

creatinine value of greater than 3 mg/dL, factors reflecting more severe renal injury and failure of other systems.

According to recent study even mild decreases in renal function are now recognized as being associated with worse patient outcomes. Inthis is evidenced by a study of contrast nephropathy subjects whose serum creatinine rise by at least 20% to 3 mg/dL and was associated with a greater than fivefold increase in mortality even after adjustment for confounding factors . Although it was not clear with the use of bivarient levels of renal function, to what extent the relationship is influenced by the subjects with more extreme depression in function. A study of 6000 general ICU patients found significant association between early degrees of AKI as assessed by wifle score and mortality. Eventhough with the use of renal replacement therapy, mortality remains high as compared with those with stable independent renal function.

In addition to its clinical importance , ARF prolongs hospital stays and is associated with more increased medical expenditure. The U.S. cost of treated AKI per Quality Adjusted year life (QAYL) was estimated in 1999 to be $50,000 per QAYL , a level that raises concerns regarding the cost effectiveness of an treatment. In a more recent analysis of long-term

outcomes of ICU patients who had recovered from renal failure quality survival was poor—20 QAYLs per 100 patient-years in the first year afterdischarge. However the patients health satisfaction was not significantly different from that of the general .

There are many problems in the design of clinical trails that have examined the efficacy of several novel therapeutic interventions on the acute kidney injury . assessment of the effect of treatment interventions in AKI is complicated by in accurately define the onset and resolution of AKI . In addition, most clinical trials of AKI in humans have limited because of an imbalance in the randomization of risk factors among controls and experimental . This problem could be dealt with by stratifying patients before randomization or ideally, by studying numbers of patients enough for adequate randomization. Therefore an accurate scoring systems is needed to classify patients enrolled in clinical trials and also to allowing physicians to make informed treatment decisions regarding medical care when the patient's condition make further intervention reasonable . The two most widely used models for critically ill patients are version 3 of the Acute Physiology and Chronic Health Evaluation (APACHE 3), version 4 of the Acute Physiology Score and

version II of the Mortality Probability Model at 48 hrs . These models were developed for severe aki patients and although APACHAE 3 may have in patients requiring renal replacement therapy, none are reliably features of outcome in the subgroup of patients with AKI. More recently, several ARF specific indices have been developed, but this indices outside the environment in which they were developed is uncertain. In general, although several of scoring systems are developed from an epidemiology and research context, they remain poor determinants of outcome in the individual patient. Finally, many human studies of ARF suffer from absence of well-defined criteria. Although the need for dialysis has been used as an end point in many trials of ARF, uniform endpoints for the initiation and continuation of dialysis has been before the study. The necessary duration of follow-up to fully capture the sequelae of an episode of AKI is not known . Follow-up of cases clearly extend beyond ICU discharge and equally beyond hospital discharge, because there is evidence that mortality rates start to stabilize after 3 months following hospital discharge.

Independent renal function is attained in most patients who survive an episode of AKI . However, 40% have subclinical functional in

glomerular filtration, tubule secretion and transport , H^+ secretion, and urinary diluenting mechanisms, and andbiobsy for glomerular or tubulointerstitial scarring . AKI is irreversible in approximately 10% of patients. It occurs because of complete cortical necrosis. It also requires long-term renal replacement therapy with dialysis or sometimes renal transplant is needed. An additional 2 to 3 % of patients suffer progressive deterioration in renal function after an initial phase, probably because of subsequent sclerosis of remnant glomeruli. According to Experimental models and of course in humans who experience one episode of AKI are at increased risk of additional episodes of AKI on subsequent exposure to further insults. It is vital to note that the subsequent predisposition to acute and chronic renal failure may become increasingly common as human life expectancy increases.

RUSSEL VIPER

It is characterized by presence of with chain of rings on the side of vertebrae and three or more chains on the back which is placed

symmetrically on both side. It is a large and thick viper

SAW SCALED VIPER

It is characterized by a coils in the shape of eight all around the body .it is the most common species causing envenomations . It is aggressive and with a sound of a saw cutting wood by rubbing its scale.

King Cobras have been found up to 18 feet long. Its hollow fangs are up to 1/2 inch long. Poison is forced through the fangs when the cobra bites.

The scaly skin glistens . Adults are yellow, green, brown, or black. The throat is light yellow or cream-colored. Juveniles are black with yellow or white bars crossing the body. The King Cobra smells using its forked tongue.

The average length is 0.9 meter. Males are longer, with proportionately longer tails. The head is flat and the neck hardly evident. The body is cylindrical. tail is tapering . The tail is short and rounded. The eyes are rather small, with rounded pupils.The head shields are normal, with no loreals. The third and fourth supraoculars touch the eyes.[2]

NON POISONOUS SNAKES :

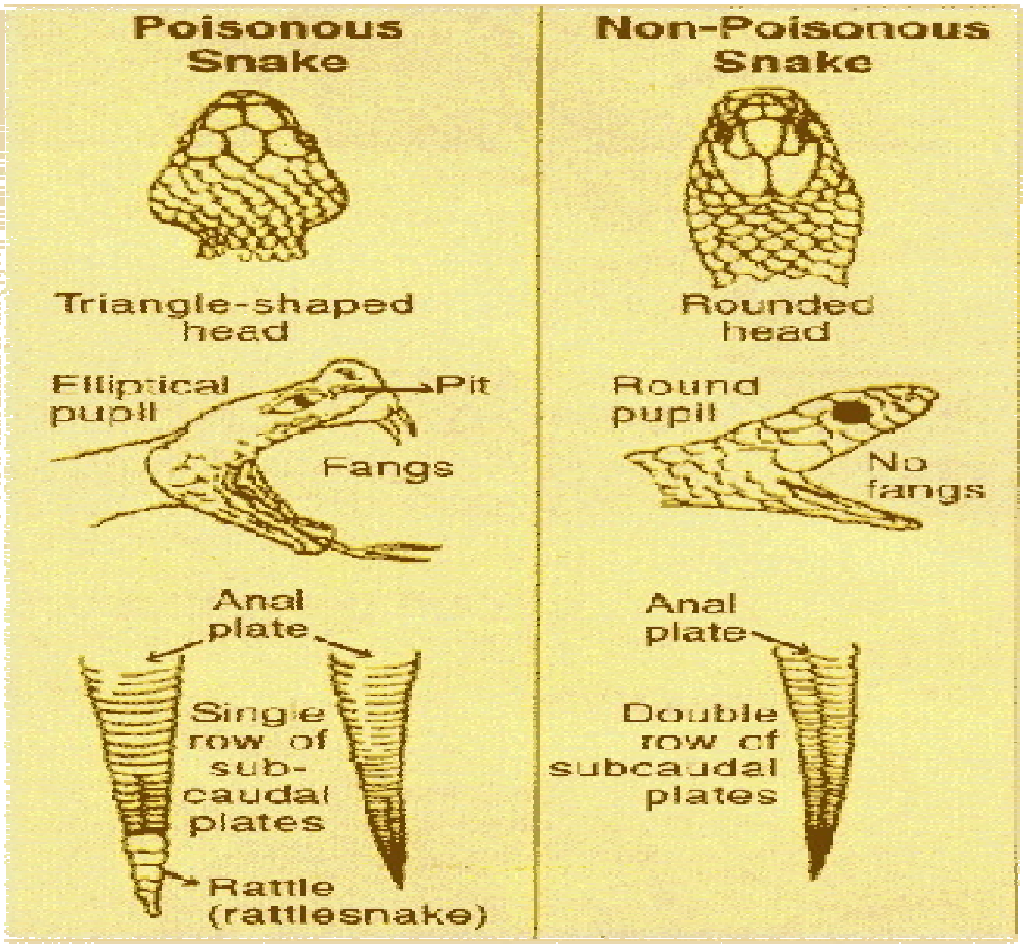
GREEN VINE SNAKE

GARTER SNAKE

TEXAS RA SNAKE

HOGNOSE SNAKE

DIFFERENTIATION OF POISONOUS AND NON POISONOUS SNAKES: :



SNAKE BITE AND ITS COMPLICATIONS:

Epidemiology:

Viperidae ,Elapidae are the two most common poisonous snakes species. Bite rates are more in common in tropical areas.it also occurs in the temperate parts of the world . the exact rate of snake bites and its envenomations and its complications including death rates are not available in Indian registry. This is because of wide way of approach and treatment including the native treatments are done in our country.

Identification :

Each snake species is identified by its characteristics structure

In case of vipers, for example russel viper it is shown by its presence of with chain of rings on the side of vertebrae[32] . three or more chains on the back .it is placed symmetrically on both side. In case of russel viper It is a large and thick viper .it coils in the shape of eight all around the body .it is the most common species causing envenomations . It is aggressive and with a sound of a saw cutting wood by rubbing its scale .the teeth has long mobile fangs that placed against the roof of the mouth. In typical snake-venom, the venom glands situated below the eye. it was connected to hollow anterior maxillary teeth by the ducts. In case of elapids and sea snakes, the fangs are small. it was in an erect position. poisioning occurs in 50% of all venomous snakebites.[33]

Dry bites means there is no envenomations on snake bites. This can occur in around 60% of the snake bites. It is common in the tropical countries.

Venoms and Clinical Manifestations.

It have multisystem effects on their patients. proteolytic enzymes causes effects like local tissue necrosis . by the same mechanism it also causes the dysregulation of the the coagulation pathway . in case of severe envenomation the enzymes released by the snake species says Hemorrhagins that cause vascular leakage. It cause both local and systemic bleeding. Some species affects the cardiac output by the direct deprresent effects of the heart. Neurotoxins act by varying mechanism . mostly it affects the peripheral nerves and the post synaptic ganglions ..Viperids and some elapids cause progressive local swelling by release of enzymes like yylauronidase. Pain, ecchymosis , and hemorrhagic bullae and serum-filled vesicles can also occur by the same mechanism.krait

cause neurologic

dysfunction .In serious bites, tissue loss can also occur . Changes in taste, mouth numbness, muscle fasciculations can also occur. Similiarly tachycardia or bradycardia, hypotension, pulmonary edema can also occur. Complications like hemorrhage , and renal dysfunction can also occur.[34, 35].some species can also cause neurologic dysfunction .tiger snakes , some cobras ,

and some viperides South American rattlesnake and some Indian Russell's viper are some of the examples . following are the neurologic dysfunction says . Ptosis, altered mental status ,respiratory paralysis. It can leads to respiratory failure and aspiration . its manifestations depends mainly on the type of species bitten and the amount of venoum injected and the duration of the delay in getting teatment. Sea snake envenomation usually causes local pain , myalgias, rhabdomyolysis .

Neurotoxicity occur. 

Treatment of Venomous Snakebite :

Airway, breathing, and circulation are the initial resuscitation measures.antivenom administration is the cornerstone of management.. extremity should be kept at approximately heart level .Splint should be apply to the bitten extremity to lessen bleeding. And it should be used only in case of non necrosis snake bites. application of poultices, ice, and electric shock should be avoided incising wounds should not be done.Applying suction to the bite should be avoided. These are ineffective and can local tissue damage.. Tourniquet use can result in amputation and loss of function ..In case of neurotoxic species pressure-immobilization can be used .

HOSPITAL MANAGEMENT:

The level of swelling in the bitten extremity should be marked . limb circumferences measured every 15 min. This is done to assess the progression of cellulitis. patient should be closely monitored for vital signs, cardiac function , oxygen saturation, urine output . Swelling size should be measured for assessing the progression of local envenomation. The extremity should be positioned at approximately heart level. Large-bore IV access in the extremities should be established. Pooling of blood in the pulmonary and splanchnic vascular beds is the reason for early hypotension .this can also complicated by systemic bleeding. This can be managed by 20–40 mL/kg of isotonic saline[36] . 5% albumin may be given . It should be given when patients fail to respond to saline infusion. Investigation includes .It includes complete blood count. renal and hepatic function. coagulation studies. testing of urine for blood or myoglobin. The 20-min whole-blood clotting test is a reliable diagnosis of coagulopathy. Dopamine should be added only after aggressive volume resuscitation. Antivenomadministration, central venous or pulmonary arterial pressures can be helpful in such cases.

Typing and cross-matching should be done .Blood should be drawn

immediately fo this. And laboratory evaluation as soon as possible. Caution of any vessel puncture in the setting of coagulopathy,if it happen it requires direct-pressure application . After giving antivenom therapy , laboratory values should be rechecked every 6 h. complete blood count and coagulation studies should be repeated every hour . 20 minutes whole blood clotting test is very useful. This consists of a few milliliters of fresh blood are placed in a glass tube. undisturbed for 20 min and should checked whether clot has formed. Coagulopathy is diagnosedif blood not clotted in 20 minutes. Patients bitten by neurutoxic snakes should be watched carefully .Evidence of cranial nerve dysfunction should b assessed. It includes difficulty in swallowing , respiratory insufficiency. It may require securing of the airway by mechanical ventillation.

Arterial blood gas studies, electrocardiography, and chest radiography can also warranted. Administration antivenom is the vital in the management of venomous snakebite .resulting in . Antivenoms are produced by the injection of venoms into animals . horses or sheepare commonly used. Antivenom antibodies is monospecific. Useful only for a particular snake species . there are also polyspecific antivenoms covering several medically important species in the region. but it cross-protection against snake species are rarely occur..After animals develop antibodies to the venoms, their serum is removed and the antibodies are isolated . And antivenom preparatiois made. It includes varying degrees of digestion and purification of the IgG molecules. The basic principle of antivenom theraphy is thefollowimg. Binding of circulating venom components before they can attach to target tissues. So antivenom selection must be specific for the offending snake. Because , it may lead to unnecessary complications if the antivenom chosen does not contain antibodies to that snake's venom components. Indications for antivenom administration include any evidence of systemic envenomation followed by laboratory abnormalities and local findings like soft tissue swelling crossing a joint .

After the bite of an unidentified snake some species cause mild edema at the bite site . care must be used because the saliva of some relatively harmless species can cause this. The efficacy of antivenoms in preventing tissue damage caused by necrotizing venoms is very less. It may be impossible to prevent necrosis completely. Because venom components bind to local tissues very quickly—before antivenom administration can be instituted. So antivenom administration should begin as soon as possible. This can prevent further tissue damage and systemic effects. In such bites, antivenoms are useless and potentially harmful. If there is evidence of neurotoxicity in this case antivenom administration after the bites of neurotoxic elapids is indicated . otherwise is not indicated. Whenever possible, it is advisable for treating physicians to seek advice from experts. Opininon obtained regarding indications and dosing of antivenom In case of viper bites, antivenom administration generally should be continued as needed until the victim shows definite improvement . Patient should have stabilized vital signs, reduced pain, restored coagulation. It is difficult to reverse neurotoxicity even with antivenom once neurotoxicity is occurred. In such cases, maintainece on mechanical ventilation is the ideal one.

Use of any heterologous serum product causes risk of acuteanaphylaxis . delayed-type hypersensitivity reactions can also occur. Skin testing for potential allergy is insensitive and should not warranted. Around 50 % of the patients can exposed to the complications of the anti snake ve om and its complications . it is prevented by the pretreatment with IV antihistamine. Antivenom dose to be administered should be diluted in an appropriate volume of crystalloid . It is remembered that antivenom should be given only by the IV route. Epinephrine at a dose of 0.01

mg/kg, up to 0.04mg/kg can be given prophylactic subcutaneous or intramuscular dose . Modest expansion of the patient's intravascular volume with crystalloids may prevent adverse reactions. Epinephrine and the infusion should be started slowly. The physician at the bedside during the initial period to look for the first signs of an acute reaction. Because serious complications like bronchospasm or acute cardiovascular collapse can be preventable .. The decision to administer further antivenom to a stabilized patient generally should be based on clinical condition. Antivenom is effective only in reversing active venom toxicity; . The rate of infusion can be increased gradually in the absence of a reaction. The dose has been administered over period of 1 hour .But the complications like renal failure and compartment syndrome and neuropartalysis can only be recover after some period of time with effective suppoertive measures.

In case if the patient develops an acute reaction to antivenom, the dose should be diluted further in isotonic saline.If the patient develops reaction to the antivenom the infusion should be temporarily stopped it should be treated immediately treated with IM epinephrine and IV antihistamines. Steroids may also be useful. Once the reaction is controlled, and restarted

as soon as possible. Concomitant IV infusion of epinephrine may be needed. The patient must be monitored very closely. An intensive care setting, during such therapy.

Blood products are rarely necessary in the management of an envenomated patient. A drop in platelet count hematocrit and removal of coagulation factors can occurs in some of the snake species. Care should be undertaken in giving blood samples to the coaguiopathy as it aggrevates or cause DIC in case of consumptive coagulopathy.

Rhabdomyolysis and hemolysis should be managed in different ways . Victims who develop acute renal failure should be evaluated by a nephrologist. They may require peritoneal dialysis or hemodialysis . Renal failure in snake bites is usually is due to acute tubular necrosis and is frequently reversible. Acetylcholinesterase inhibitors like neostigmine can be useful .It is useful neurologic dysfunction after snakebite should give a trial. If they If they respond, additional doses of long-acting neostigmine can be given. If bilateral cortical necrosis occurs, the prognosis for renal recovery is poor. Renal transplantation may be necessary in this case.

Edrophonium and neostigmine may cause neurologic improvement. It

occurs in patients bitten by snakes with postsynaptic neurotoxins. Care is required to prevent aspiration in case of neostigmine administration.Care of the bite wound includes application of a dry, sterile dressing . Splninting of the extremity with padding between the digits can be done. Once the administration of antivenom has been initiated, the extremity should be elevated above heart level to prevent edema. Tetanus toxoid should be given as appropriate. Prophylactic antibiotics are generally unnecessary because the incidence of secondary infection is low. In some regions, secondary bacterial infection is more common . In these regions, prophylactic antibiotics are used commonly. Pain control should be achieved with acetaminophen or narcotic analgesics. NSAIDSs should be avoided because of their effects on blood clotting.

Manysnakeenvenomations involve subcutaneous deposition of venom. But some venom can be injected more deeply into muscle compartments. If swelling in the bitten extremity raises concern that subfascial muscle edema , the ICP remains high . in such case a surgical consultation for possible fasciotomy should be obtained. Although fasciotomy actually may worsen myonecrosis in some cases. compartmental decompression is still required in case to preserve nerve function. . In such cases

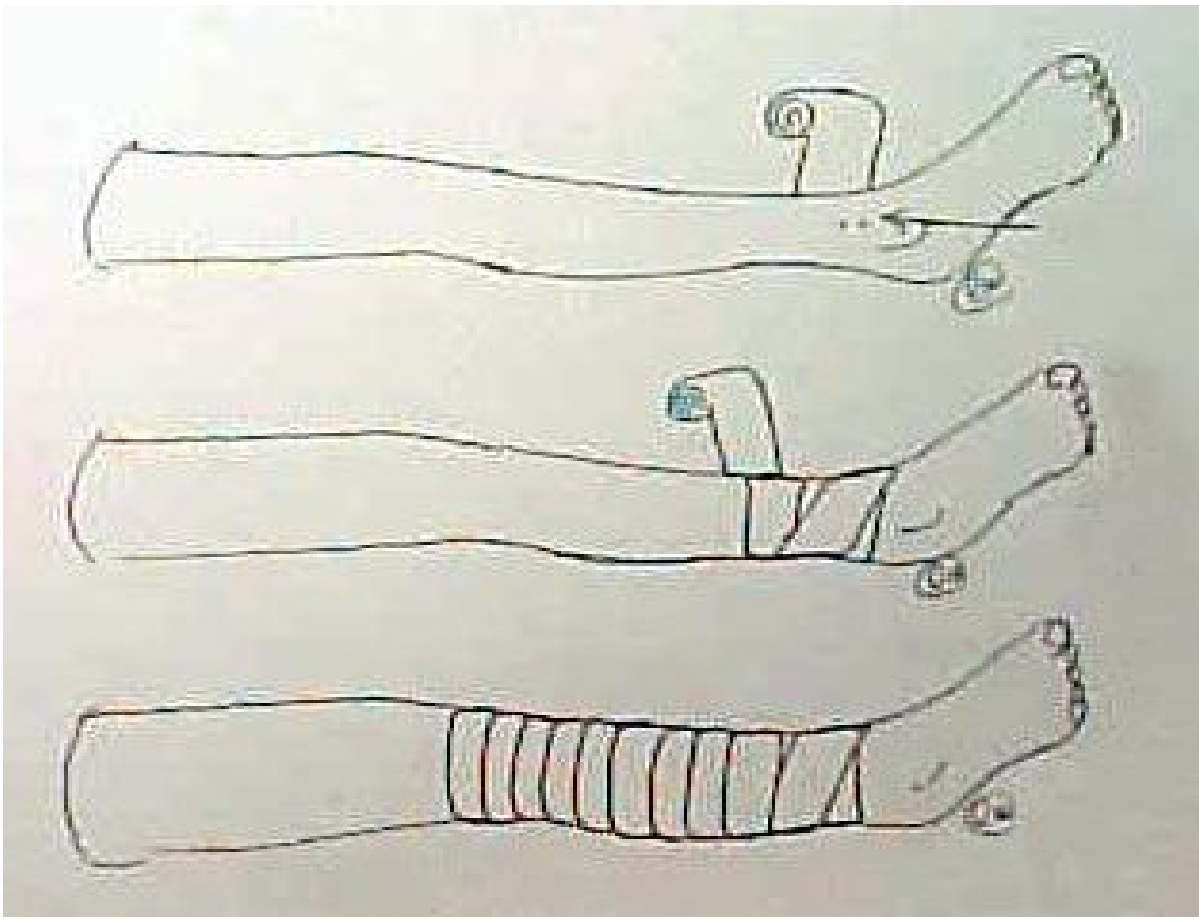
intracompartmental pressures (ICPs) should be checked. A dose of IV mannitol (1 g/kg) can be given in an effort to reduce muscle edema. The incidence of muscle-compartment syndrome is very low after snakebite. Wound care in the days after the bite may require careful debridement .Remove the necrotic tissue once coagulation has been restored.

In case of serum-filled vesicles or hemorrhagic blebs it should be should be left undisturbed. If ruptured, they should be debrided with sterile technique. Any debridement of damaged muscle should be conservative..

Mortality and morbidity:.

Snakes responsible for large numbers of deaths include cobras , carpet and saw-scaled vipers , Russell's vipers and some large African vipers. tropical rattlesnakes can also cause mortality.

The mortality rates for venomous snakebite are low in areas with rapid access to medical care. If antivenoms facillites are available it is far less.The incidence of morbidity is comparably minimal . Morbidity may be due to muscle, nerve, or vascular injury . Such morbidity can have devastating consequences for victims in the developing world .



METHODOLOGY

SOURCE OF DATA :

Snake bite patients who admitted in thanjavur medical college during the period of January to August 2014 who also fulfills the inclusion and exclusion criteria

METHOD OF COLLECTION OF DATA :

Sample size : 50 persons

Sampling method : simple random sampling .

INCLUSION CRITERIA :

1. Snake bite of all species\
2. Persons of all ages.

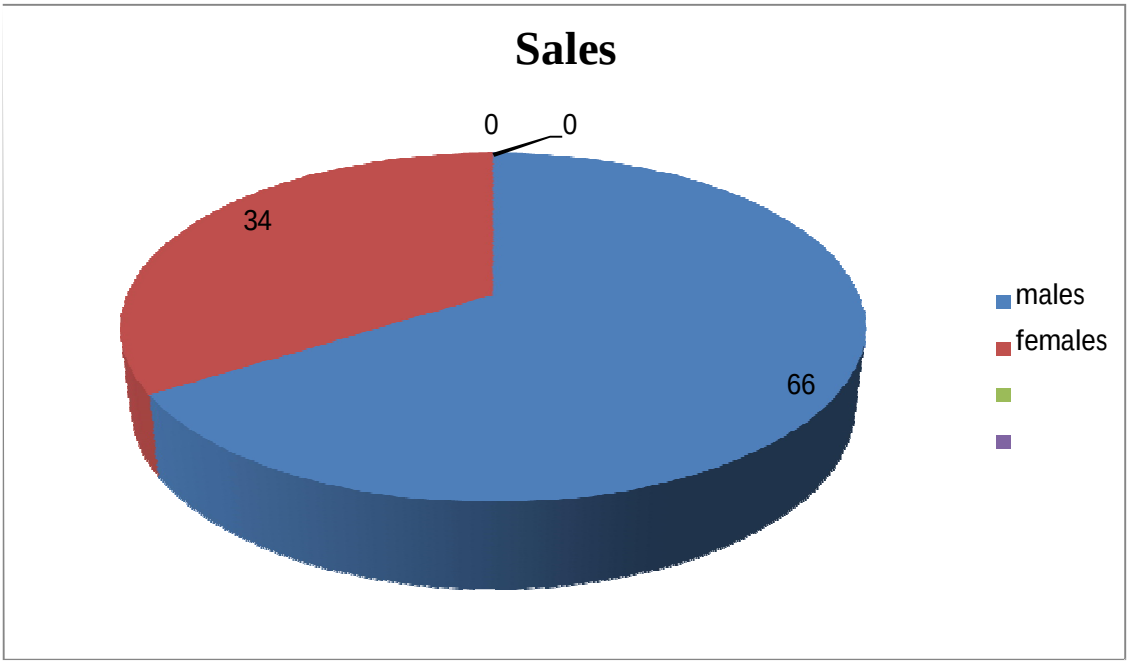
EXCLUSION CRITERIA :

1. Chronic kidney diseases
2. Unknown bite

RESULTS :

Male : female ratio

Sex	No of patients	Percentage
Male	33	66
Female	17	34

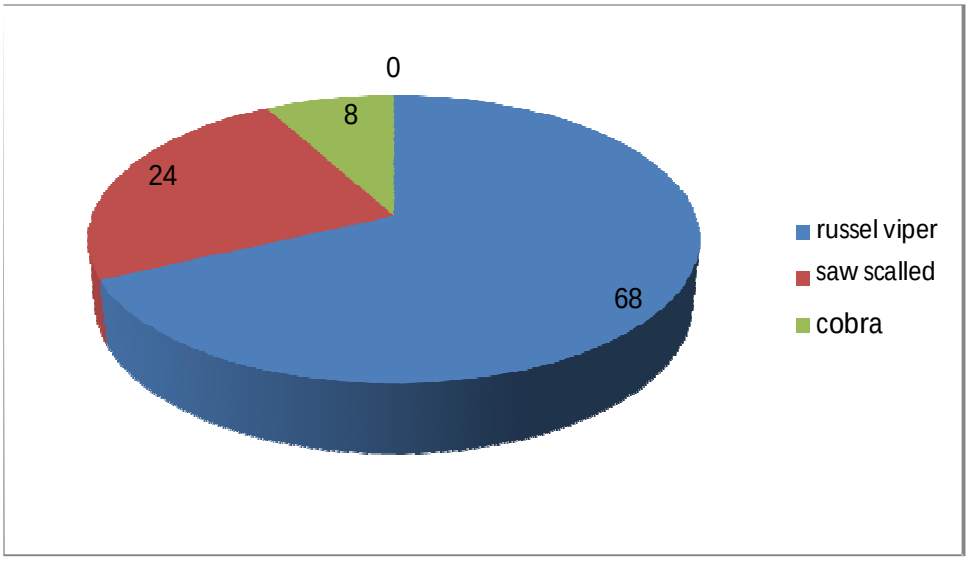


It is known from the above inference that the males are more prone for the snake bite induced acute kidney injury in percept of environment interest. It may be due to the fact of local culture also

SPECIES OF SNAKE :

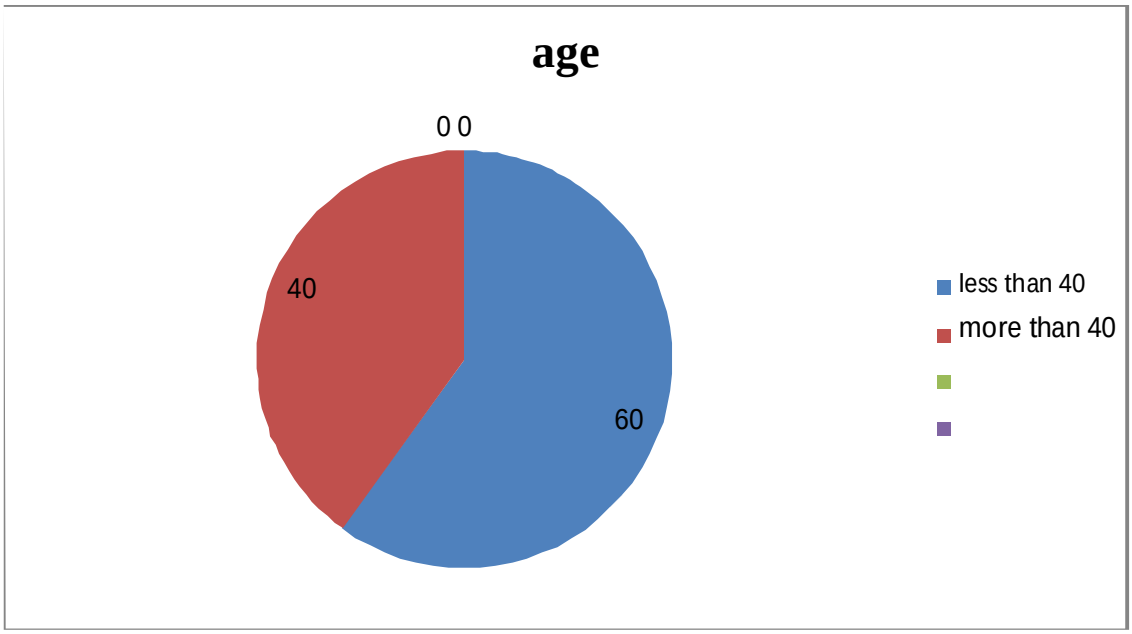
sno	Type of snake	No of patients	percentage
1	Saw scaled	12	24
2	Russel viper	34	68
3	Cobra	4	8

Inference from the above table, it was concluded that the incidence of acute kidney injury was high with the russel viper followed by saw scaled viper and cobra accounts for only few incidence



COMPARISON OF AGE < 40 and > 40

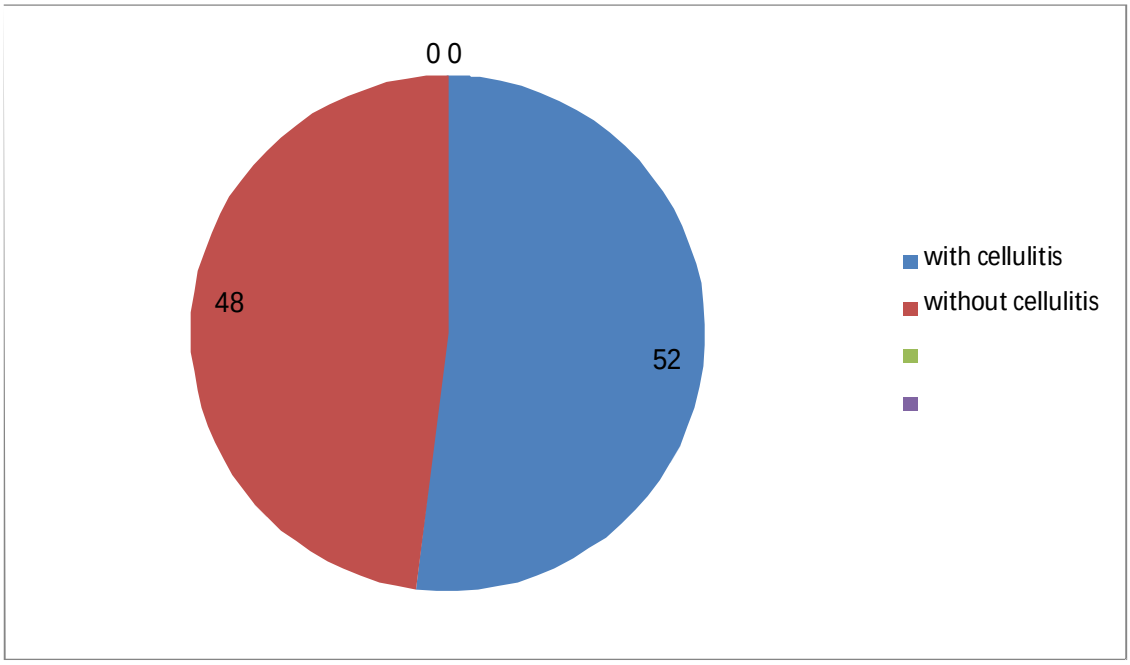
Age	No of patients	percentage	
Less than 40	20	40	
More than 40	30	60	



From the above table it was inference that the more than 40 years of age are more prone for AKI in comparison with patients aged less than 40 years

PERCENTAGE OF CELLULITIS IN AKI:

sno	Cellulitis	No of patients	percentage
1	present	26	52
2	absent	24	48



From the above table it was inference that the 52 % percent of AKI patients is having underlying cellulitis as a cofactor and 48 % not

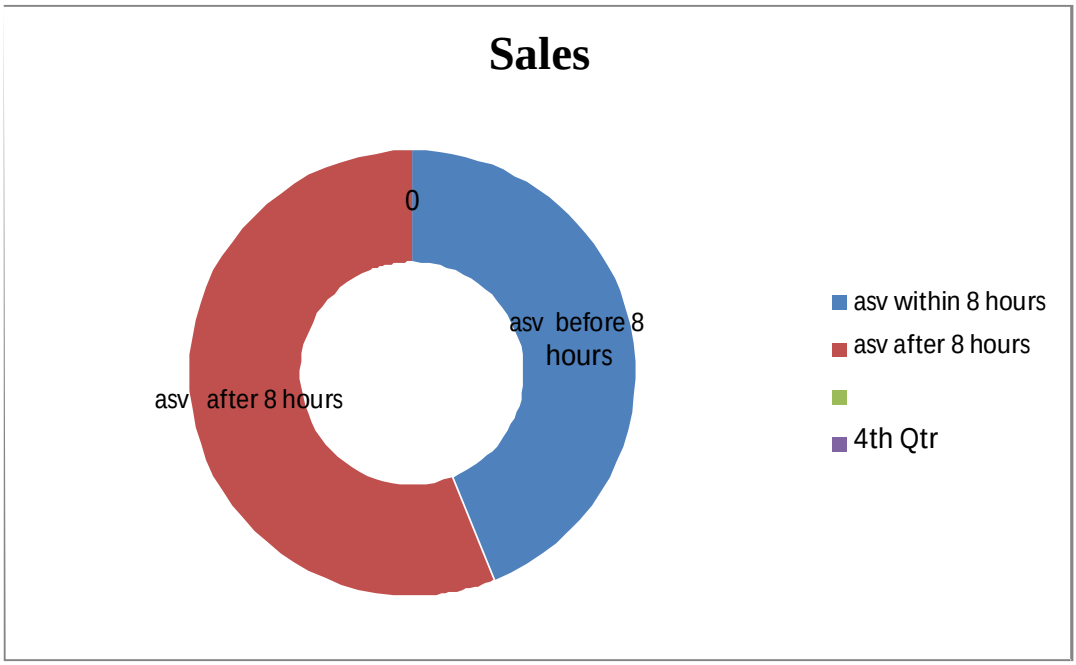
PERCENTAGE OF COAGULOPATHY IN AKI :

	coagulopathy	Percentage
Present	50	100
absent	0	0

TIME OF 1ST DOSE OF ASV ADMINISTRATION :

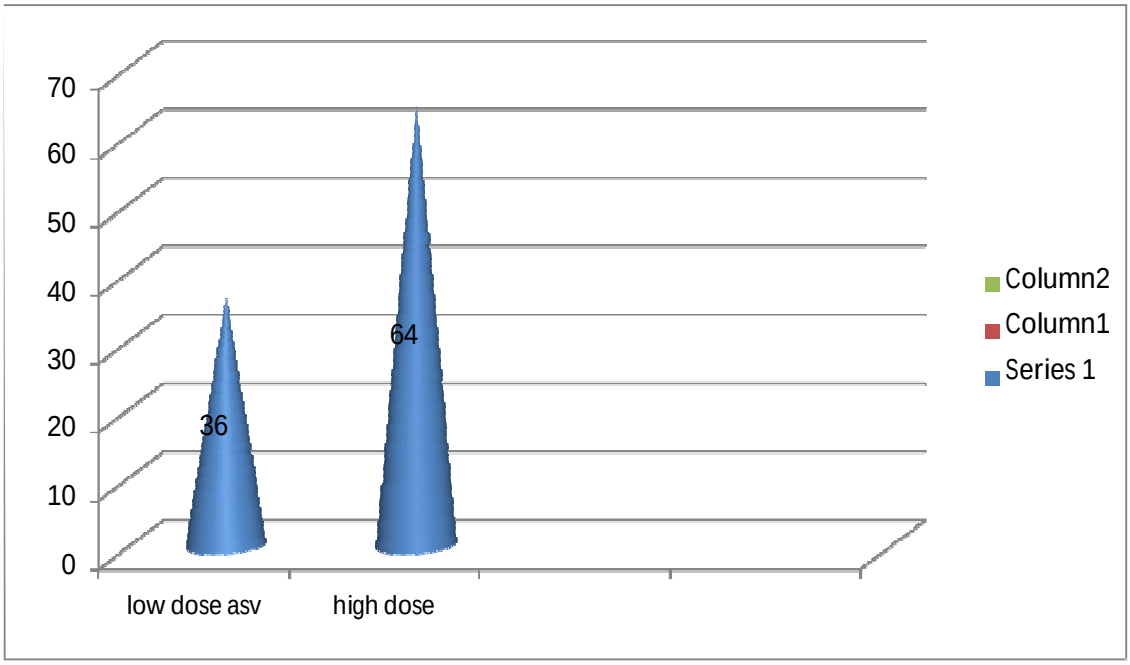
Asv first dose	No of patients	percentage
Within 8 hours	28	56
More than 8 hours	22	44

From the table it was inferenced that the time interval of 1st dose of asv whether it was within 8 hours or more than 8 hours have no big relation to the occurrence of acute kidney injury


Sales
0
asv before 8 hours
asv after 8 hours
asv within 8 hours
asv after 8 hours
4th Qtr

PERCENTAGE OF LOW DOSE VS HIGH DOSE ASV CAUSING AKI

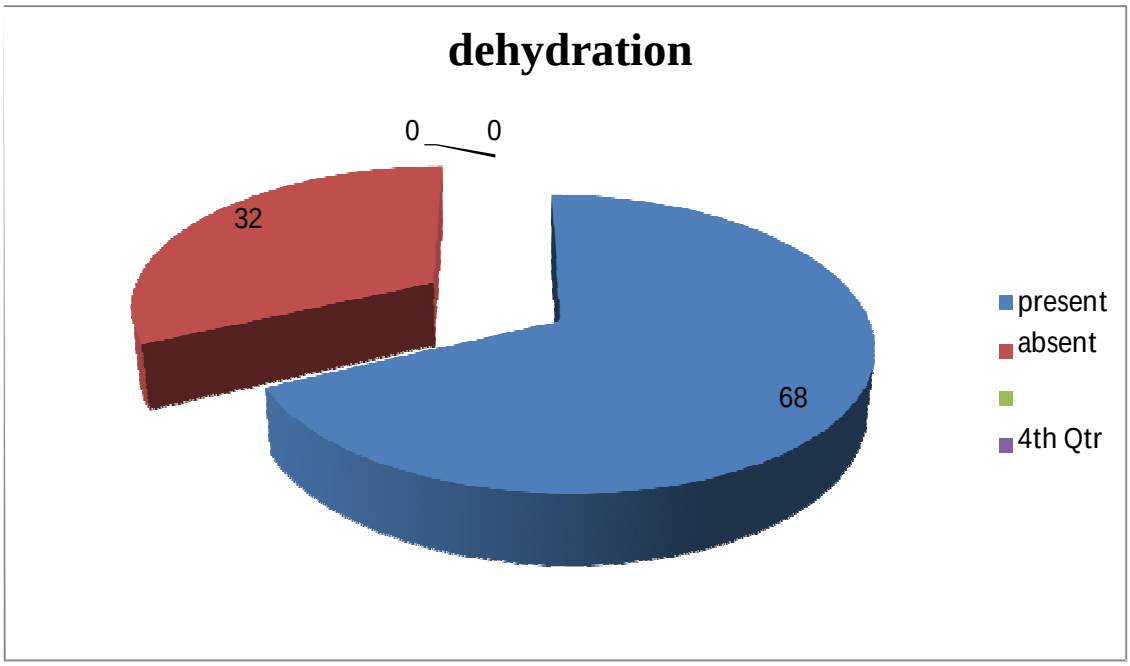
asv	No of patients	percentage
Low dose	18	36
High dose	32	64



From the table it was inferenced that the high dose of ASV is not protective under AKI

PERCENTAGE OF PATIENTS WITH DEHYDRATION PRONE FOR AKI :

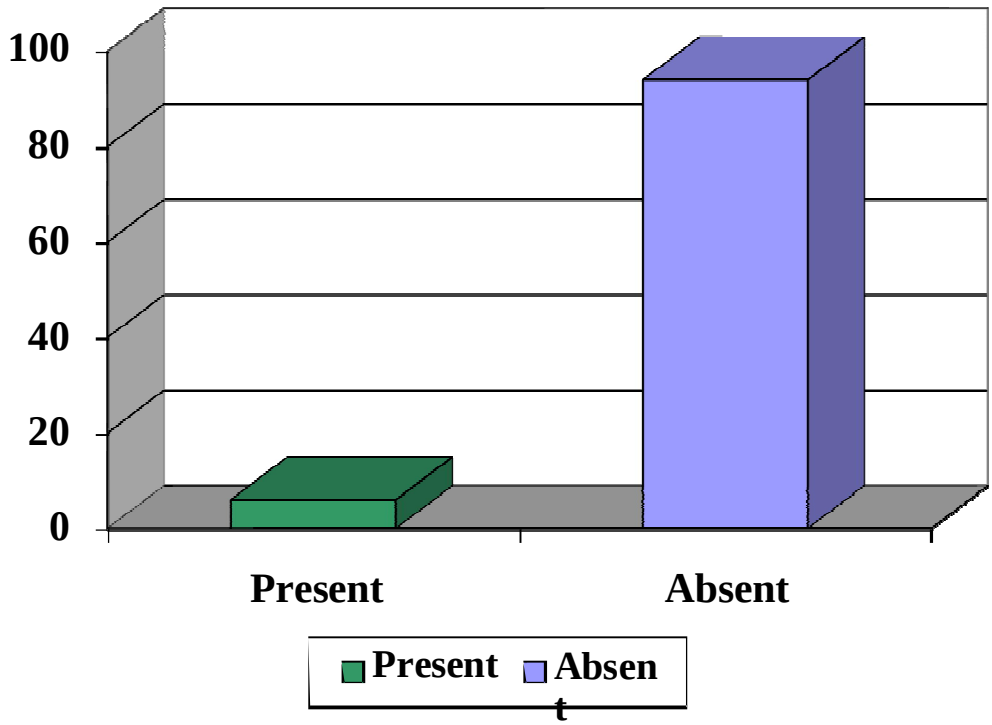
Dehydration	No of patients	percentage
Present	34	68
absent	16	32



From the above table it was inferenced that the dehydration is strong predictors of AKI

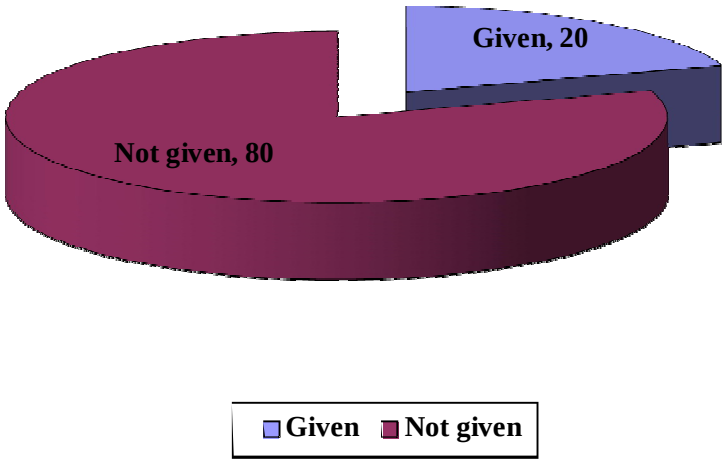
PERCENTAGE OF PATIENTS WITH OR WITHOUT RISKFACTORS PRONE FOR AKI :

Risk factors	No of patients	percentage
present	3	6
absent	47	94



PERCENTAGE OF PATIENTS WITH INTENSIVE CARE AND WITHOUT INTENSIVE CAUSING AKI :

Intensive care	No of patients	percentage
given	10	20
Not given	40	80



DISCUSSION :

This is a retrospective analysis study of factors favouring acute kidney injury in snake bite patients

50 patients are take into account who are bitten by snake and was admitted in thanjavur medical college who also fulfills the inclusion and exclusion criteria

10 factors are take into account which are thought to be a reason behind causing acute kidney inury says

1. Age
2. Sex
3. Species of snake
4. Cellulitis
5. Coagulopathy
6. Time interval of first dose of asv
7. High dose vs low dose asv
8. Dehydration
9. Risk factors for kidney injury

10.Intensive care vs general care

SAMPLING TYPE :

Simple random sampling

SUBJECTS : 50

1.INCLUSION CRITERIA :

1. Snake bite of all species
2. Persons of all ages.

EXCLUSION CRITERIA :

3. Chronic kidney diseases
4. Unknown bite

The results of the study are analyzed and was plotted as a stataistical diagrams and the inference were noted .

1. ANALYSIS OF AGE OF PATIENTS WHO ARE PRONE FOR ACUTE KIDNEY INJURY :

As we know increasing age is an independent risk factor for kidney injury

Age	No of patients	percentage	
Less than 40	20	40	
More than 40	30	60	

Patients more than age 40 are more prone for acute kidney injury . associated comorbide illness like prehypertension, impaiered glucose tolerance and diabetic mellitus and hypertension can also be a cause in patients aged more than 40 years

Analysis of type of species of snake that are more prone for AKI :

AS WE KNOW there are many types of snake spcies which may be poisonous or nonpoisonous

There are also species which are neurotoxic , hemotoxic or myotoxic

The following table shows the comparison of three common type of species of snake that are more prone for acute kidney injury

sno	Type of snake	No of patients	percentage
1	Saw scaled	12	24
2	Russel viper	34	68
3	cobra	4	8

From the above table , it clearly shows that the russel viper is a species which most commonly associated with acute kidney injury in a term of percentage says 68 % followed by saw scaled viper in 24 % and copra rarely causes acute AKI

Analysis of cellulitis as a risk factor for acute kidney injury :

In this study only cellulitis that cross the joint space is taken into account as a cellulitis that causing the AKI

Localized cellulitis is common in any insect bite that may be due to a local anaphylasis reaction :

sno	Cellulitis	No of patients	percentage
1	Present	26	52
2	Absent	24	48

According to our study cellulitis is found to be an moderate risk factor in term of causing acute kidney injury.

More than fifty percent of patients are more prone for AKI in comparison with patients without cellilitis

Coagulopathy and acute kidney injury :

Surprisingly our study has found that all 50 patients in the studry are having underlying coagulopathy . so from it we can come to a conclusion that coagulopathy and AKI have strong dependent mechanism. This

mechanism may be strong influence on causing renal tubular acidosis and subsequent acute kidney injury .

	coagulopathy	*Percentage*
Present	50	*100*
absent	0	*0*

The table shows all 50 patients in the study are having coagulopathy as a underlying risk factors which is statistically very significant.

LOW DOSE VS HIGH DOSE ASV :

IT WAS A CONCERN ALL OVER THE world whether the high dose of asv can prevent the complications of snake bites. But our study shows it was not the high dose asv that prevents the complications. So even large number of patients in oue study aeround 32 were given large dose of asv , there is no reduction in the incidence of AKI.

asv	No of patients	percentage
Low dose	18	36
High dose	32	64

THE above table clearly shows that the high dose of asv is not protects against acute kidney injury. In oyr study we have taken 8 vbials of asv as a cutoff point out for high dose vs low dose asv.

Time interval OF 1ST DOSE OF ASV GIVEN :

IT WAS believed from the long time that the administration of asv as early as possible can prevent the complications of snake bites.

But our study disbelieve this fact that the initiation of early asv is not protective against complications of snake bites. So it was inference from this fact may be t dose or amount of snake venom can cause this complications .

Asv first dose	No of patients	percentage
Within 8 hours	28	56
More than 8 hours	22	44

So it is clear from the above table that the early institution of asv is not protective against the complication of snake bite especially AKI.

DEHYDRATION/VOMITING :

It was a recent interest in the field of medicine whether the dehydration caused by vomiting , anaphylactic shock to asv or by anyother cause can cause acute kidney injury as because dehydration is a known independent cause of any kidney insult.

Dehydration	No of patients	percentage
Present	34	68
absent	16	32

So from the above observation it was clear that the dehydration is the major factor contributing to the development of acute kidney injury in snake bite patients

So correction or preventing the dehydration can largely prevents the development of acute kidney injury patients .

INTENSIVE CARE :

It was done to find out is there any major change in occurrence of AKI if the patients were given intensive care set up.

Because less number of patients were included in intensive care group in our study because in our study place most of the snake bite patients and patients developed acute kidney injury were managed in general ward itself.

Intensive care	No of patients	percentage
given	10	20
Not given	40	80

So it is very difficult to interperatefom this study taking account of this small number of patients about intensive care.

But we can say from the finding of large number pf patients were managed in general ward itself..we can come to a conclusion indirectly that the there wont be any major change in intensive or general ward.

COEXIXTING RISK FACTOR FOR KIDNEY INJURY :

This includes pre hypertension, impaired glucose tolerance, diabetic mellitus, hypertension .

As we know all this factors can prone kidney injury this was also analysed with snake bite patients,

But in our study most of the patients don't have any coexixting risk factors so it helps our study to purely analyse other factors in causing AKI.

Risk factors	No of patients	percentage
present	3	6
absent	47	94

It is obvious that any coexisting risk factors can influence the incidence of acute kidney injury.

Comparison with other studies :

1. Jayantha paul et.al studies on early prediction of acute kidney injury of snake bite patients correlates better with our study in perceptive of coagulopathy and hypotension.

CONCLUSION :

1. COAGULOPATHY HAS BEEN OBSERVED IN 100% OF SUBJECTS PRONED FOR ACUTE KIDNEY INJURY.
2. DEHYDRATION HAS BEEN SECOND MOST FAVOURABLE FACTORS IN PREDISPOSING ACUTE KIDNEY INJURY IN SNAKE BITE PATIENTS .
3. HIGH DOSE ASV IS NOT PREVENTING OR REDUCING THE INCIDENCE OF ACUTE KIDNEY INJURY IN SNAKE BITE PATIENTS .
4. EARLY INSTITUTION OF ASV IS ALSO NOT FOUND TO BE REDUCING THE INCIDENCE OF ACUTE KIDNEY INJURY IN SNAKE BITE PATIENTS.
5. RUSSEL VIPER SNAKE BITE WERE THE COMMON SPECIES ACCOUNTING FOR 68% OF CASES OF ACUTE KIDNEY INJURY FOLLOWED BY SAW SCALED VIPER OF 24 % AND LEAST BY COBRA ACCOUNTS FOR 8 %.

6. PROGREESIVE CELLULITIS IS NOT FOUND TO BE A MAJOR FACTOR IN CAUSING ACUTE KIDNEY INJURY.
7. AGE ,SEX,COEXISTING RISK FACTOR, INTENSIVE CARE ARE FOUND TO BE NOT A MAJOR FACTORS INFLUENCING THE OCCURRENCE OF ACUTE KIDNEY INJURY IN SNAKE BITE PATIENTS.

LIMITATIONS OF THE STUDY :

1. NOT INCLUDED SUBJECTS WITH SIMILAR UNDERYING FACTORS AND ARE NOT DEVELOPED ACUTE KIDNEY INJURY.
2. PROGNOSIS OF ACUTE KIDNEY INJURY IS NOT ASSESED.

BIBLIOGRAPHY

1. Brady HR, Singer GG: Acute renal failure. *Lancet* 1995; 346:1533-1540.

2. Klahr S, Miller SB: Acute oliguria. *N Engl J Med* 1998; 338:671-675.

3. . Liano F, Pascual J: Epidemiology of acute renal failure: a prospective, multicenter, community-based study. Madrid Acute Renal Failure Study Group. *Kidney Int* 1996; 50:811-818.

4. Blume A, Kaschina E, Unger T: Angiotensin II type 2 receptors: signalling and pathophysiological role. *Curr Opin Nephrol Hypertens* 2001; 10:239-246.

5. Hays R, Aquino A, Lee BB, et al: Captopril-induced acute renal failure in a kidney transplant recipient. *Clin Nephrol* 1983; 19:320-321.

6. Racusen LC: *The morphologic basis of acute renal failure*. In: Molitorids BA, Finn WF, ed. *Acute Renal Failure*, New York: W.B. Saunders Company; 2001:1-12.

7. Brivet F, Kleinicht D, Loirat P, et al: Acute renal failure in intensive care units—causes, outcome and prognostic factors of hospital mortality: a prospective, multicenter study. *Crit Care Med* 1996; 24:192-198.

8. Hotchkiss RS, Karl IE: The pathophysiology and treatment of sepsis. *N Engl J Med* 2003; 348:135-150.

9. Wangsiripaisan A, Gengaro PE, Edelstein CL, et al: Role of polymeric Tamm-Horsfall protein in cast formation: oligosaccharide and tubular fluid ions. *Kidney Int* 2001; 59:932-940.

10. Friedewald JJ, Rabb H: Inflammatory cells in ischemic acute renal failure. *Kidney Int* 2004; 66:486-491

11. Pallone TL, Turner MR, Edwards A, et al: Countercurrent exchange in the renal medulla. *Am J Physiol Regul Integr Comp Physiol* 2003; 284:R1153-R1175.

12. Olof P, Hellberg A, Kallskog O, et al: Red cell trapping and post-ischemic renal blood flow. Differences between the cortex, outer and inner medulla. *Kidney Int* 1991; 40:625-631.

13. Bellomo R, Ronco C, Kellum JA, et al: Acute renal failure—definition, outcome measures, animal models, fluid therapy and information technology needs: the Second International Consensus Conference of the Acute Dialysis Quality Initiative (ADQI) Group. *Crit Care* 2004; 8:R204-R212.
14. Ueda N, Kaushal GP, Shah SV: Apoptotic mechanisms in acute renal failure. *Am J Med* 2000; 108:403-415
15. Ortiz A: Nephrology forum: apoptotic regulatory proteins in renal injury. *Kidney Int* 2000; 58:467-485.
16. Patel NS, Sharples EJ, Cuzzocrea S, et al: Pretreatment with EPO reduces the injury and dysfunction caused by ischemia/reperfusion in the mouse kidney in vivo. *Kidney Int* 2004; 66:983-989
17. . Bamburg JR: Proteins of the ADF/cofilin family: essential regulators of actin dynamics. *Annu Rev Cell Dev Biol* 1999; 15:185-230.
18. Brady HR, Lieberthal W: *Acute renal failure*. In: Brenner B, ed. *The Kidney*, 6th ed. Philadelphia: WB Saunders; 1999.
19. Mattern WD, Sommers SC, Kassirer JP: Oliguric acute renal failure in malignant hypertension. *Am J Med* 1972; 52:187-197
20. Anderson RJ, Chung HM, Kluge R, et al: Hyponatremia: a prospective analysis of its epidemiology and the pathogenetic role of vasopressin. *Ann Intern Med* 1985; 102:164-168.

21. Choi PT-L, Yip G, Quionez LG, et al: Crystalloids vs. colloids in fluid resusitation. A critical review. *Crit Care Med* 1999; 27:200-210.
22. Carpenter CC, Mitra PP, Sack RB, et al: Clinical studies in Asiatic cholera II. Development of a 2:1 saline:lactate regimen. Comparison of this regimen with traditional methods of treatment. *Bull Johns Hopkins Hosp* 1966; 118:174-196.
23. Hatala R, Dinh T, Cook DJ: Once-daily aminoglycoside dosing in immunocompetent adults: a meta- analysis. *Ann Intern Med* 1996; 124:717-725.
24. Conger JD: Acute uric acid nephropathy. *Med Clin North Am* 1990; 74:859-871.
25. Marik PE, Iglesias J: Low-dose dopamine does not prevent acute renal failure in patients with septic shock and oliguria. NORASEPT II Study Investigators. *Am J Med* 1999; 107:387-390.
26. Solomon R, Werner C, Mann D, et al: Effects of saline, mannitol, and furosemide to prevent acute decreases in renal function induced by radiocontrast agents. *N Engl J Med* 1994; 331:1416-1420.
27. Fiaccadori E, Maggiore U, Giacosa R, et al: Enteral nutrition in patients with acute renal failure. *Kidney Int* 2004; 65:999-1008

28. Owen WF, Lazarus JM: *Dialytic management of acute renal failure*. In: Lazarus JM, Brenner BM, ed. *Acute Renal Failure*, 3rd ed. New York: Churchill Livingstone; 1993:487.
29. Eustace J, Heffernan A, Watson A: *Acute hemodialysis and acute peritoneal dialysis*. In: Brady HR, Wolcox CS, ed. *Therapy in Nephrology and Hypertension*, Philadelphia: W.B. Saunders; 1999:53-69.
30. Cimochowski GE, Worley E, Rutherford WE, et al: Superiority of the internal jugular over the subclavian access for temporary dialysis. *Nephron* 1990; 54:154-161.
31. Ragaller MJ, Theilen H, Koch T: Volume replacement in critically ill patients with acute renal failure. *J Am Soc Nephrol* 2001; 12(Suppl 17):S33-S39.
32. 4. Myint L, Warrell DA, Phillips RE, Tin NS, Tun PE, Maung ML. Bites by Russell's viper (Vipera russelli siamensis) in Burma. Haemostatic, vascular, and renal disturbances and response to treatment. Lancet.1985;2:1259–64. [PubMed]
33. The global burden of snakebite: A literature analysis and modelling based on regional estimates of envenoming and deathspublished 03 November 2008.
34. . Sweni S, Meenakshisundaram R, Sakthirajan R, Rajendiran C, Thirumalaikolundusubramanian P. Acute renal failure in acute poisoning: Prospective study. J Ren Care. 2011;38:22–8. [PubMed

35. Chugh KS, Sakhuja V. Renal disease caused by snake venom. In: Tu AT, editor. Handbook of Natural Toxins. New York: Marcel Dekker Inc; 1991. pp. 471–93.
36. New Delhi, India: World Health Organization, Regional Office for Southeast Asia; 2005. Guidelines for the clinical management of snake bites in the Southeast Asia region

PROFOMA

ANALYSIS OF FACTORS FAVORING ACUTE KIDNEY INJURY IN SNAKE BITE PATIENTS

NAME :

IP NO. :

AGE :

SEX:

SPECIES OF SNAKE BITE :

CELLULITIS : PRESENT / ABSENT

BLEEDING HISTORY:

VOMITING /DEHYDRATION : PRESENT / ABSENT

COMORBID ILLNESS: DIABETES/ HYPERTENSION/ OTHERS

TIME INTERVAL OF FIRST ASV: < 8 HRS / > 8 HRS

LOW DOSE VS HIGH DOSE ASV : < 8 VIALS / > 8 VIALS

INTENSIVE CRITICAL CARE: YES /NO

EXAMINATION :

PROGRESSIVE CELLULITIS PRESENT/ ABSENT

INVESTIGATIONS:

RANDOM BLOOD SUGAR:

UREA:

CREATININE:

BT:

CT:

PT:

INR:

USG ABDOMEN:

S.No.	IP No.	AGE	SEX	SPECIES	CELLULITIS	COAGULOPATHY	TIME INTERVAL OF 1ST DOSE ASV	LOW DOSE VS HIGH DOSE	DEHYDRATION	RISK FACTOR	INTENSIVE CARE	UREA	CREATININE	USG ABDOMEN
1	34607	43	M	VIPER	NO	YES	3 HRS	LOW	YES	NO	YES	118	4.1	AKI
2	35812	40	F	UNKNOWN	NO	YES	4 HRS	HIGH	YES	NO	NO	84	3.1	AKI
3	36416	28	M	SAW SCALED VIPER	NO	YES	4 HRS	HIGH	YES	NO	NO	78	2.8	AKI
4	36812	56	F	UNKNOWN	YES	YES	5 HRS	HIGH	YES	NO	NO	62	2	AKI
5	36900	54	M	VIPER	NO	YES	3 HRS	LOW	NO	NO	NO	96	3.1	AKI
6	40104	38	F	UNKNOWN	NO	YES	14 HRS	HIGH	NO	NO	YES	112	3.8	AKI
7	41111	46	F	UNKNOWN	YES	YES	5 HRS	LOW	YES	NO	NO	90	3	AKI
8	41284	42	M	RUSSEL/VIPER	NO	YES	8 HRS	LOW	YES	NO	NO	84	3	AKI
9	46824	38	M	VIPER	YES	YES	3 HRS	HIGH	YES	NO	NO	100	3.2	AKI
10	46920	45	M	UNKNOWN	YES	YES	8 HRS	HIGH	NO	YES	YES	92	2.8	AKI
11	47000	37	M	VIPER	YES	YES	14 HRS	LOW	YES	NO	YES	78	2.6	AKI
12	52100	52	F	SAW SCALED VIPER	YES	YES	5 HRS	HIGH	NO	NO	NO	114	3.6	AKI
13	52380	46	M	UNKNOWN	YES	YES	11 HRS	HIGH	YES	NO	NO	94	3.2	AKI
14	54830	34	M	UNKNOWN	YES	YES	3 HRS	LOW	YES	NO	NO	84	2.8	AKI
15	55823	51	F	RUSSEL/VIPER	NO	YES	8 HRS	HIGH	NO	NO	NO	68	2.2	AKI
16	57960	37	F	VIPER	NO	YES	7 HRS	HIGH	YES	NO	NO	70	2.8	AKI
17	58380	45	F	RUSSEL/VIPER	YES	YES	11 HRS	LOW	NO	NO	NO	112	3.8	AKI
18	59480	34	M	UNKNOWN	YES	YES	5 HRS	LOW	YES	NO	YES	68	2.4	AKI
19	60308	38	M	UNKNOWN	NO	YES	12 HRS	HIGH	YES	NO	NO	86	2.8	AKI
20	61832	48	M	SAW SCALED VIPER	YES	YES	12 HRS	HIGH	YES	YES	NO	90	3.2	AKI
21	62216	30	M	UNKNOWN	NO	YES	7 HRS	LOW	YES	NO	NO	80	2.6	AKI
22	63128	51	F	UNKNOWN	NO	YES	6 HRS	HIGH	NO	NO	NO	94	3.6	AKI
23	65122	36	M	UNKNOWN	NO	YES	6 HRS	LOW	YES	NO	NO	68	1.8	AKI

24	67000	54	M	RUSSEL/VIPER	YES	YES	14 HRS	LOW	NO	NO	NO	74	2	AKI
25	68124	51	M	UNKNOWN	NO	YES	8 HRS	HIGH	NO	NO	YES	96	2.8	AKI
26	69012	40	F	VIPER	NO	YES	3 HRS	HIGH	YES	NO	NO	112	3.6	AKI
27	70120	26	M	VIPER	NO	YES	4 HRS	HIGH	YES	NO	NO	80	2	AKI
28	70280	60	M	UNKNOWN	YES	YES	7 HRS	HIGH	NO	NO	NO	110	3.2	AKI
29	72328	46	M	VIPER	YES	YES	5 HRS	HIGH	YES	NO	NO	86	3	AKI
30	73128	45	M	RUSSEL/VIPER	YES	YES	11 HRS	HIGH	YES	NO	YES	84	2.4	AKI
31	75105	57	M	VIPER	YES	YES	7 HRS	HIGH	YES	NO	NO	76	2.4	AKI
32	77128	37	F	UNKNOWN	NO	YES	5 HRS	HIGH	NO	NO	NO	112	5.2	AKI
33	77256	33	M	UNKNOWN	YES	YES	4 HRS	LOW	NO	NO	NO	68	1.8	AKI
34	77860	55	F	VIPER	YES	YES	7 HRS	LOW	NO	NO	YES	80	3	AKI
35	79058	38	M	RUSSEL/VIPER	YES	YES	10 HRS	LOW	YES	NO	NO	100	4	AKI
36	80185	26	M	UNKNOWN	YES	YES	8 HRS	HIGH	YES	NO	NO	100	3.8	AKI
37	80282	43	M	RUSSEL/VIPER	YES	YES	6 HRS	LOW	YES	NO	NO	78	2.6	AKI
38	81428	50	F	RUSSEL/VIPER	NO	YES	5 HRS	LOW	YES	NO	NO	82	2.8	AKI
39	82210	38	F	VIPER	NO	YES	12 HRS	HIGH	NO	NO	NO	68	2.2	AKI
40	82410	41	M	UNKNOWN	NO	YES	7 HRS	HIGH	YES	NO	NO	80	2.8	AKI
41	83280	42	M	UNKNOWN	YES	YES	8 HRS	HIGH	YES	NO	NO	92	2.8	AKI
42	83418	52	M	UNKNOWN	YES	YES	10 HRS	HIGH	YES	NO	NO	78	2.2	AKI
43	84860	48	M	VIPER	NO	YES	12 HRS	HIGH	YES	NO	NO	86	2.6	AKI
44	85098	31	M	RUSSEL/VIPER	NO	YES	6 HRS	HIGH	NO	NO	NO	100	3.2	AKI
45	86092	28	F	UNKNOWN	NO	YES	5 HRS	LOW	NO	NO	YES	92	2.8	AKI
46	87121	56	F	RUSSEL/VIPER	YES	YES	4 HRS	LOW	YES	YES	NO	98	3	AKI
47	87434	34	M	RUSSEL/VIPER	YES	YES	11 HRS	HIGH	YES	NO	NO	64	1.8	AKI
48	87521	38	M	VIPER	YES	YES	12 HRS	HIGH	YES	NO	NO	72	2	AKI
49	87548	42	M	UNKNOWN	NO	YES	8 HRS	HIGH	YES	NO	YES	76	2.2	AKI

50	88380	44	F	UNKNOWN	NO	YES	7 HRS	HIGH	YES	NO	NO	84	2.8	AKI

ABBREVIATIONS

AKI- acute kidney injury

RTA- Renal tubular acidosis

ASV- Anti snake venom

TT- TETANUS TOXOID

PD- peritoneal dialysis

HD- hemodialysis

PT- prothrombin time

CT- clotting time

DIC- disseminated intravascular coagulation

aPTT- activated partial thromboplastin time

ATN- Acute tubular necrosis

GFR- glomerular filtration rate

CONSENT FORM

I _____ hereby give consent to participate in the study conducted by **DR.E.DEYAGARASAN ,** post graduate in department of internal medicine ,Thanjavur medical college & Hospital, Thanjavur – 613001 and to use my personal clinical data and result of investigation for the purpose of analysis and to study the nature of disease. I also give consent for further investigations.

Place :

Date : Signature of participant